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The Journal of Thoracic Surgery

VOL. 5 APRIL, 1936 NO. 4

Original Communications

MANAGEMENT OF BILATERAL CAVERNOUS PULMONARY TUBERCULOSIS*

BILATERAL CASEOUS PNEUMONIC PULMONARY TUBERCULOSIS

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NEW YORK, N. Y.

INTRODUCTION

THE term "bilateral caseous pneumonic pulmonary tuberculosis" designates the ulcerative form of the disease, characteristic of which is the tuberculous cavity. Patients with cavities in one lung alone and with strictly proliferative and productive lesions in the contralateral lung are not classified as bilateral caseous pneumonic cases. As we have shown elsewhere,[1, 2] these cases, following pneumothorax or thoracoplasty upon the ulcerated lung, behave as strictly unilateral cases. In the present study we shall deal exclusively with cases presenting cavities in both lungs.

The management of these advanced bilateral cases of pulmonary tuberculosis constitutes today the most important problem of the treatment of this disease. Notwithstanding the considerable progress accomplished during the last decade by the surgical collapse of the tuberculous lung in unilateral cases, very little has been accomplished for the treatment of bilateral cases. Attention has been focused upon the unilateral and curable cases. Rather rigid operative indications have been formulated limiting the choice of operative cases to a very restricted variety and number of patients. Considerable fear of discrediting surgical treatment led to excessive limitations of operative indications.

*From the Departments of Thoracic Surgery and Tuberculosis of Metropolitan and Sea View Hospitals of the City of New York.

Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery at New York City, June 3, 4, 5, 1935.

This was certainly a wise measure a few years ago when incomplete knowledge of the fundamentals of pathologic physiology of tuberculosis of the lungs and faulty technic were followed by prohibitive operative mortality. In order to overcome the strong resistance of patients and physicians to the novelty of surgical treatment of this disease, it was necessary to produce a high percentage of good results and to avoid catastrophes. Conditions, however, have changed, and the merits of surgical collapse have gained general recognition. Perfection of surgical technic and better knowledge of the etiology, prevention and treatment of postoperative complications have reduced the postoperative mortality to almost the vanishing point and have tremendously increased the number of permanent clinical cures.

The strict discrimination between good, fair and bad operative cases, as formulated by Archibald,[3] and others only a few years back, appears obsolete today. We have become much bolder in our operative indications with increasingly gratifying results.

The problem changes completely in bilateral ulcerative pulmonary tuberculosis. The number of attempts at surgical treatment of these cases, published so far, is negligible when compared with the number of unilateral cases surgically treated. Sporadic cases have been published of late in increasing numbers. Combinations of all known surgical procedures have been applied along these lines.[4] A considerable number of cases of bilateral pneumothorax completed by closed pneumonolysis,[5] unilateral or even bilateral phrenicectomies, combined with pneumothorax,[6] unilateral or bilateral, apicolysis with packing or plombe combined with other procedures;[7] thoracoplasties in conjunction with pneumothorax or other methods of collapse in the contralateral lung,[8] or even a restricted number of bilateral thoracoplasties[9, 10] with good results have been reported.

These, however, have been only isolated efforts which have been considered by the ultraconservative wing of phthisiologists rather as happy acrobatic surgery. In fact, there has been no painstaking persistent and systematized effort to solve the problem of the management of far advanced bilateral ulcerative pulmonary tuberculosis considered as a whole. It seems that the consensus of opinion of phthsiologists and surgeons has been and still is that these patients were beyond surgical help and that the only reasonable management was to assure them of shelter, symptomatic treatment, and humane care. A great deal has been done to preserve the commonwealth from these patients but very little has been done to cure them.

During the last three years we have systematically studied this fascinating problem. In fact, in the two municipal institutions for tuberculous patients of the City of New York, namely, Sea View and the tuberculosis division of Metropolitan Hospital, composed of 2,200 beds, 65 per cent of all cases entrusted to our medical and surgical care are

represented by these advanced bilateral patients. As do most of the municipal and state institutions for tuberculous patients, these hospitals represent the end of the trail of far advanced tuberculous patients who are transferred from other charitable or private institutions to die in peace.

The results so far obtained by our studies upon the surgical management of these cases have been very encouraging. They have been more favorable than we had anticipated. We have come to believe that these results are due not so much to perfected operative technic as to a new working hypothesis upon which our efforts were based. This hypothesis has induced us to change completely not only our modus operandi but also the operative indications and the rules upon which the choice of cases had been based. The theory upon which a new perception of the whole problem was based was the logical conclusion of our previous investigation, both experimental and clinical, upon the pathologic physiology, chemistry, and mechanics of pulmonary tuberculosis, and more especially upon the biology of tuberculous cavities. Therefore, the thoughts expressed in this paper and the results presented should not be taken and judged as happy surgical attempts due to perfected technic; they should rather be appraised as a whole system, elaborated by the close cooperation of the surgeon with the phthsiologist, and based upon new theoretical conceptions originated in the laboratory, verified by the study of pathologic specimens, and finally corroborated by its application to the patient.

For a better exposition of our subject we shall first discuss the value of the operative indications in bilateral cases which are accepted today. We shall then briefly present the mechanics, pathologic physiology, and pathology of ulcerative tuberculous lesions, that is, cavities upon which our conception was based. Following this, we shall formulate our working theory and the operative indications derived from it and terminate with the technic used and the results thus far obtained.

PRESENT-DAY OPINION CONCERNING OPERATIVE INDICATIONS IN BILATERAL CAVITIES

The operative indications for bilateral cases so far accepted are so vague and so restrictive that they become, for practical purposes, quite prohibitive. The most comprehensive list of indications we know of is the one formulated by Jessen in 1934.[10] We shall, therefore, limit our discussion to these indications, which are generally accepted today.

Jessen considers that the operability of these patients depends upon the following conditions: (1) the general immunity, (2) the tertiary allergic character of the cavities, (3) the absence of even latent myocarditis, (4) the relative rigidity of the mediastinum, (5) the capacity of retraction not yet ended, (6) the absence of extrapulmonary foci

(intestine, kidneys, except larynx), and (7) the general disposition of the patient, physical and mental.

Simple perusal of these conditions shows their vagueness. It is obvious that instead of clarifying the problem they further obscure it by the introduction of more unknown and indefinite factors.

In fact, we know very little if anything about the first condition, which concerns the degree of immunity of the patient to tuberculosis, and even less about determining it. Is it a steady or a variable factor? Can it be modified and by what means? What is its relation to tuberculous allergy, if any? Not one of these questions has thus far been satisfactorily answered. How, then, is it possible to derive from this unknown factor of "immunity to tuberculosis" a positive or negative indication for surgical treatment?

The second condition of Jessen deals with cavities of tertiary allergy in which surgical treatment would be indicated, whereas it would not in secondary allergy cavities. This factor is no less indefinite than immunity. In fact, Jessen himself admits that "these cavities often merge, they can change in a reversible sense" or even that "they can persist one beside the other." This being the case, how can we accept the statement of this author that "each of these two groups of cavities corresponds to a certain state of immunity and allows different possibilities of endogenous propagation" and consequently different therapeutic indications? It is obvious that this condition is perfectly useless as a criterion of the operability of bilateral or even of unilateral cases with cavities.

The third requisite of Jessen is absence of myocarditis, even latent; we do not think that myocarditis is of frequent occurrence in tuberculous patients. A number of patients who died of so-called postoperative cardiac failures had a perfectly healthy heart before operation, and death was due to anoxemia rather than to a latent myocarditis. Therefore, we believe that unless we deal with a definitely diseased myocardium, proved such by electrocardiographic examination, we are justified in not considering patients unfit for surgical treatment because of the possibility of latent myocarditis.

The same can be said for the fourth consideration of Jessen, namely, absence of rigidity of the mediastinum. We do not believe that absence of rigidity of the mediastinum constitutes a serious contraindication to surgical collapse. In fact, when the collapse of a diseased lobe or lung by pneumothorax is not hindered by the presence of adhesions, the lung collapses completely though pleural pressure remaining negative, and independently of the mobility of the mediastinum. Likewise, in thoracoplastic collapse, mobility of the mediastinum is of no importance; in fact, when cavities in the collapsed lung remain open, this is due to mechanical causes which will be developed below and not to the mobility or rigidity of the mediastinum.

The fifth condition of Jessen for fitness of surgical treatment is that "the capacity of retraction (of the diseased parenchyma) be not yet ended." Again we deal with a very vague condition. It is impossible to predict what course an apical lesion will follow, and we all agree that a cavity should be collapsed without any procrastination; we all have had patients with small or large cavities, located in the midst of retracting or completely retracted parenchyma, in whom widespread extensions of the disease were produced after hemoptysis. These bronchogenic extensions often occur without previous suspicion of the impending catastrophe. Therefore, we believe that no time should be lost in the discussion of such niceties as the above mentioned conditions of Jessen. The sixth condition, concerning the absence of extrapulmonary lesion, is a concrete and definite one, if it be not extended to all extrapulmonary lesions. In fact, intestinal and laryngeal tuberculosis, if not far advanced, do not constitute contraindications. On the contrary, the best treatment for these complications is closure of the cavities. Unilateral renal tuberculosis is not a formal contraindication. In a patient of our series, nephrectomy was performed first and later thoracoplasty. Another patient had vertebral fusion for Pott's disease, and six months later thoracoplasty was performed. Both of these patients are today well and bacilli free. Intestinal tuberculosis is improved after collapse of the tuberculous lung.

The seventh condition of Jessen concerns the general disposition of the patient in a physical and mental sense. By this broad term the author probably understands such complicating circumstances as hyperthyroidism, diabetes, pregnancy, advanced amyloid degeneration, etc. As for the mental disposition, unless the patient is insane, there is no formal contraindication to surgical collapse. If the patient is opposed to the operation, the best way to convince him about its effectiveness is to place him in an institution provided with a well-functioning surgical service, because the example of the other patients is the best means for clearing up the erroneous mental disposition toward pneumothorax or thoracoplasty. A few years ago it was extremely difficult to obtain consent of patients for collapse therapy in our institutions; today they ask for surgical treatment.

We have discussed rather extensively the operative indications as formulated by Jessen, as they represent the prevailing opinions.

We do not believe that it is possible to advance the problem of management of bilateral cases by following the indications discussed above. The foundations upon which these premises are based are much too vague and indefinite. Therefore, a firmer groundwork should be sought. It is our contention that this groundwork exists. It is represented by the pathologic physiology, mechanics, and biology of tuberculous cavities.

PATHOLOGIC PHYSIOLOGY, MECHANICS, AND BIOLOGY OF TUBERCULOUS CAVITIES

However true it may be that tuberculosis is a general disease, according to Ranke, and that isolated pulmonary tuberculosis is found only in the lungs when they are extracted from the body, it is none the less true that in at least 50 per cent of our cases, pulmonary tuberculosis, in a clinical sense, behaves as if it were a strictly pulmonary disease. Furthermore, most often the disease is localized in one lung or even in one lobe and may remain there for a considerable length of time. All collapse treatment is based upon this property of pulmonary tuberculosis. On the other hand, the conception presented recently by one of us,[11, 12, 13] according to which the closure of cavities is due to the narrowing and obliteration of their communicating bronchi, opens up new pathways of attack of the whole problem of treatment of pulmonary tuberculosis. Besides, it gives us a rational explanation of the action of collapse treatment and points to the direction that our efforts should follow.

We have not yet found any means of directly attacking these bronchi and producing their closure. We are able, however, to obtain the same result by producing their collapse indirectly through mechanical collapse of the diseased lung. It was shown that closure of the draining bronchi causes absorption of the air entrapped in the cavities and brings about their atelectatic collapse and stops the passage of bacilli into the bronchial tree, thus preventing further extension of the disease; furthermore, it was proved that disappearance of oxygen from the cavity arrests the growth of the strictly aerobic tubercle bacilli and the production of toxic products of their metabolism. No one can support the thesis that closure of cavities means the cure of the disease, since tubercle bacilli may remain alive in them for a long time. But so long as the disease remains arrested, latent and without any clinical symptoms, we can consider that the patient is no more phthisic than any one who has been primarily infected but never showed any other manifestations during the entire length of his life. Pathologic and clinical evidence are in perfect agreement on this point.

This being the case, it is difficult to understand the tendency of phthsiologists to base indications upon niceties of immunity, resistance, general disposition, and mental attitude instead of upon the fundamental and easily proved properties of tuberculous cavities which, after all, represent the disease itself in malignant pulmonary tuberculosis.

But if by closing the cavities in unilateral tuberculosis we can arrest the disease, it is not impossible to obtain the same in bilateral and far-advanced cases. This has already been proved by the successes of bilateral pneumothorax on patients in whom successful selective collapse was obtained, leaving sufficient healthy parenchyma for insuring the oxygen metabolic needs of the organism.

Thus the question which becomes of paramount importance is not so much the degree of destruction of the lung produced by the disease and the number and size of cavities but rather the topographic distribution of the lesions. In other words, a patient with almost complete destruction of both upper lobes is more amenable to treatment than a patient with smaller cavities and lesser total destruction of the pulmonary parenchyma but in whom the disease is distributed to all lobes. In fact, in the first case after complete selective collapse of both upper lobes the patient still has at his disposal the two lower lobes, that is, more than one-half of his original lung parenchyma. In the second case the remaining respiratory field may not be sufficient for insuring adequate oxygenation even at rest.

Keith[14] has shown that when strong exercises are avoided, one-tenth of our lung is perfectly compatible with prolonged life. We have repeatedly had the opportunity to verify the opinion of Keith in our cases, as will be shown later. Thus what really counts is not so much the amount of diseased lung as the amount of healthy lung. Theoretically a patient with three-fourths of his lung diseased can still be rescued provided that the diseased parts can be efficiently collapsed.

Another well-known fact explains the often amazing results which can be obtained in apparently doomed patients. Following successful collapse of the cavities of one lung, a marked improvement is often noted in the contralateral lung. We believe that this may be due to the arrest of production of tuberculin in the collapsed cavities and therefore to the decrease of perifocal infiltration at the site of the lesions in the contralateral lung. Be this as it may, it remains definitely proved that by the selective collapse of the lesions of one side a bilateral case can be transformed into a unilateral one and thereafter treated as such.

WORKING HYPOTHESIS

We have thus come to formulate the following statement, which has served as our working hyposthesis: "No bilateral case should be considered as hopeless and unsuitable for collapse treatment because of the fact alone that it is bilateral; even if only one lobe is still healthy, treatment should be instituted." No one can foresee with any degree of accuracy what will be the further evolution of the case.

The application of this conception in our work allowed us to fix the operative indications more precisely.

The stumblingblock in collapse treatment in bilateral cases is the frequent flaring up of the disease in the contralateral lung following collapse of one side. This is especially observed in these cases following thoracoplasty. It is reasonable to consider that the sudden collapse of one apex and the postoperative retention of bronchial exudate produce a considerable limitation of the respiratory field. This

limitation may be and really is only transient; within a few days after operation, when normal means of defense of the lung have been reestablished, this excessive reduction of the respiratory area is decreased and remains confined only to the mechanically collapsed apex. But in the meantime an excessive vicarious activity has been imposed upon the contralateral lung often followed by rapid progression and extension of the disease on that side. This unfortunate complication did occur often in our first cases and almost discouraged us from further attempts.

The solution to this problem was suggested by the follow-up study of our unilateral cases in which thoracoplasty was performed. In a small percentage of these cases, spread of the disease appeared in the contralateral side. In examining the previous radiograms of these cases we found that as a rule a small lesion was present on the contralateral side, which was overlooked or was not taken into consideration because of its apparent harmlessness. It occurred then to one of us (G. G. O.) that in the future a diagnostic pneumothorax should be attempted upon the contralateral lung in all cases in which there was suspicion that a small cavity was present. It is known that often in such cases pneumothorax renders visible a cavity otherwise invisible in radiograms or fluoroscopic examinations.

In numerous patients, by this procedure, previously unsuspected small cavities were visualized. A step further was taken in these cases; it was decided to keep on with pneumothorax while thoracoplasty was performed on the other side. The results were very gratifying. In fact, even a small selective pneumothorax seemed to prevent the noxious effects of the vicarious hyperactivity of the contralateral lung mentioned above.

Encouraged by these results, we applied the same procedure in all bilateral cases. Pneumothorax was first induced on the more affected side; if a good selective collapse was obtained on this side, pneumothorax was induced on the other side. If the collapse on either side was unsuccessful because of the presence of adhesions, every effort was made to complete it by internal closed pneumonolysis (Jacobaeus' operation). In these cases pneumonolysis was pushed to its extreme limits. Since the only hope of these patients was the production of an adequate selective collapse by pneumothorax on at least one side, we inaugurated the technic of gradual section of the adhesions in stages. The results thus obtained were so favorable that we have become gradually bolder, and we have modified the technic and the instrumentation of this procedure in order to adapt it to these new requirements.* In a number of cases bilateral selective collapse was obtained following staged pneumonolyses on both sides.

*This technic and the modified instruments will be described in a forthcoming paper.

In a second series of cases, pneumothorax could be induced on only one side; in these cases thoracoplasty was performed on the other side.

In a third class of our cases pneumothorax could not be done on either side. In these patients, who have a more severe prognosis, bilateral thoracoplastic collapse was attempted.

In a limited number of cases apicolysis with plombe on one side and pneumothorax in the other, or bilateral apicolysis with plombe was performed. Detailed analysis of the procedures used and the results obtained in our 238 cases will follow below.

This series of cases in which 40 to 60 per cent clinical cures were obtained (see results) has produced a complete change in our ideas upon the operability of these patients who otherwise were doomed to certain death. Therefore, we came to revise the indications accepted thus far and to formulate new rules based exclusively upon the topographic distribution of cavities.

THERAPEUTIC INDICATIONS

Even fairly advanced cases of laryngeal or intestinal tuberculosis do not contraindicate collapse treatment by pneumothorax or thoracoplasty.

In the presence of extrapulmonary tuberculosis, operability is determined as in unilateral cases; so, when bone, joint or kidney tuberculosis are present, these lesions are treated independently of the pulmonary localization.

Likewise, when other conditions complicate pulmonary tuberculosis, the indications for collapse treatment are the same as in ordinary unilateral cases; amyloidosis, hyperthyroidism and diabetes are the most frequent of these conditions. Amyloidosis in the absence of amyloid nephrosis does not constitute a formal contraindication. Hyperthyroidism requires previous correction of this disease by thyroidectomy. Diabetes, when it can be controlled, does not preclude surgical collapse treatment of the diseased lungs.

One must never lose sight of the fact that bilateral pulmonary tuberculosis is such a desperate condition that the important consideration in our ultimate results is not the mortality but whether the number of patients saved from death can reasonably justify our efforts. It is our contention that it does.

According to the extension and distribution of the disease in bilateral cases we have recognized five groups:

1. Cavities in one lung with suspicious lesions in the other lung.
2. Cavities located in the apices, on both sides.
3. Cavities in the apex on one side, with extensive lesions in the other lung.
4. Cavities in the upper lobe on one side and in the base on the other side.
5. Extensive bilateral distribution of cavities.

Cavities on One Side With Suspicious Lesions on the Other Side

When pneumothorax is possible on the diseased side, the indications for the other side vary with the evolution of the disease; if a cavity develops, the case will pass to one of the groups of bilateral cases and will be treated accordingly. It is different when pneumothorax has not been possible in the diseased lung and thoracoplasty was therefore advised. In these cases if a most careful radiographic and fluoroscopic examination cannot inform us with certainty whether the contralateral suspicious lesion is a cavity or not, we induce what we call a diagnostic pneumothorax. As a rule, in such cases an existing cavity, however small, becomes more visible after pneumothorax. If no cavity is disclosed, the air is allowed to be absorbed, and we proceed with thoracoplasty on the diseased side. Conversely, when a cavity is thus shown, pneumothorax is maintained during the completion of thoracoplasty on the diseased side.

Bilateral Apical Cavities

The indications in this group are relatively simple; pneumothorax should be attempted on the more affected side. If successful selective apical collapse is obtained, the healthy lower lobe is allowed to reexpand and pneumothorax is attempted on the other side without any loss of time. When adhesions do not allow successful selective pneumothorax, they should be cut by closed pneumonolysis without delay. It is dangerous to use positive intrapleural pressures in these cases in order to obtain more adequate collapse. Besides the unnecessary loss of time, we expose the patients to spreads of the disease because of excessive reduction of the respiratory field following collapse of the healthy pulmonary parenchyma. Likewise, we are opposed to phrenicectomies and phrenicotomies for two reasons. In the first place, this method gives only about 20 per cent closure of cavities, it is capricious, and often its results are only temporary; in the second place, because in bilateral cases we must strive to preserve the functional integrity of all and any portion of healthy parenchyma. As long as the mobility of the chest is unimpaired, paralysis of the diaphragm is of little importance. It is different when thoracoplasty is performed; the impairment of the mobility of this hemothorax changes the diaphragm from an accessory to the principal respiratory muscle of that side. Since it is not possible to know in advance whether ultimately thoracoplasty will or will not be necessary on one side, it is unwise to jeopardize the function of the healthy lower lobe. In fact, when successful collapse of the upper lobe is obtained, the lower lobe is allowed to reexpand while the diseased upper lobe is maintained collapsed. This technic renders possible the increase of the vital capacity and allows the induction of pneumothorax on the contralateral side. Again, when adhesions prevent an adequate selective

collapse of the upper lobe, they should be sectioned by internal pneumonolysis. We have followed this technic in eight cases which appeared completely hopeless. At present five of these cases are arrested as will be shown below.

Indications become more complicated when pneumothorax cannot be induced on one side; in these cases it should be tried on the other side. If pneumothorax is successful or is successfully completed by closed pneumonolysis, the lower lobe on the pneumothorax side is allowed to reexpand and a selective apical collapse is maintained while thoracoplasty in one or several stages is performed upon the other side.

When induction of pneumothorax is impossible on either side or when the character of adhesions preclude a successful Jacobaeus procedure, thoracoplasty should be performed on both sides. In these cases the prognosis is more serious because of the greater frequency of postoperative extension of the disease. At any rate, it is the only chance for the patient, and in a number of cases the results obtained were excellent. In four of our seven cases, bacilli free sputum was obtained.

Cavities in the Upper Lobe of One Side and Extensive Ulcerative Lesion in the Other Lung

In this group the same line of reasoning is applied. Pneumothorax is attempted for the more diseased lung. If successful, or successfully completed by a Jacobaeus operation, pneumothorax is induced on the other side. When pneumothorax is impossible on one side, thoracoplasty is performed while pneumothorax is maintained on the other side. Indications for bilateral thoracoplasty in these cases when pneumothorax fails in both lungs depend upon and are regulated by the amount of healthy and adequately functioning lung which can be preserved after the diseased parts have been collapsed. It is often amazing how well patients can fare with a vital capacity as low as 900 c.c. This again shows the importance of avoiding routine phrenic nerve interruptions. Even temporary interruptions are not justified because it is seldom that the function of the diaphragm is completely restored after regeneration of the crushed or simply sectioned nerve. In fact, regeneration of the muscular diaphragm, the fibers of which have degenerated during the prolonged interruption of their motor and trophic nervous stimulations, is far from being qualitatively complete: on the other hand, its quantitative regeneration depends upon the number of nerve fibers which were integrally regenerated. This decrease of tonus explains why a previously paralyzed diaphragm, which has recovered its normal movements, may, following a moderate increase of intrapleural pressures (coughing, sneezing, etc.), be displaced during expiration downward while the healthy diaphragm moves to the opposite direction. This well-known phenomenon shows

that the temporarily paralyzed diaphragm often becomes partially but permanently impaired in its motility and tonus even following crushing of the phrenic nerve. This partial impairment may be sufficient for jeopardizing adequate and efficient ventilation of the remaining healthy lower lobe.

Cavities in the Upper Lobe on One Side and the Lower Lobe on the Other

In these cases operative indications are limited by the fact that only pneumothorax can be used on the side of basal lesions because no adequate thoracoplastic collapse of the lower lobe can be successful unless the whole lung is collapsed. These cases are the only ones in which we believe that phrenicectomy or crushing may find their indications. In our cases, however, we were not successful in producing definite closure of basal cavities with phrenic nerve interruption. It is obvious that upper thoracoplasty on the other side in case of failure of selective apical pneumothorax is indicated, theoretically at least, only when the basal cavities of the other side are closed. Because of the relative rarity of this type of bilateral lesions our experience with that group is not sufficient to warrant definite conclusions. We shall only venture the hypothesis that when the apical lesion is markedly predominant we may expect that its efficient collapse may cause the closure of the contralateral basal lesions. It is only a hypothesis which may be good to follow in suitable cases. After all, in these cases we have the choice between a problematic treatment and certain death with no treatment at all. Greater experience with these patients may modify our present conceptions.

Advanced Bilateral Ulcerative Lesions

Reasonably no surgical collapse should be attempted with these cases. However, not seldom we see in our surgical conferences patients who judging from their radiograms appear absolutely desperate; but, when we see the patient, we are literally amazed to see a man or a woman who appears perfectly healthy or hardly touched by the disease. Repeatedly we have felt morally obliged to do something for these patients. Conditions were explained to them and they, as a rule, were eager to have something done. Of course, in the majority of cases we have failed. Also, however, we have obtained a number of spectacular results which have encouraged us to make further trials, and we have often been compensated for our failures.

OTHER METHODS OF COLLAPSE

So far we have considered only two procedures of collapse, pneumothorax and thoracoplasty. Pneumothorax has taken a preponderant place in our work because of the important increase in the numbers of collapse successfully completed by the modified technic of intrapleural pneumonolysis (65 per cent).

There is, however, a number of cases in which thoracoplasty does not seem feasible. Excessive emphysema in the contralateral lung, advanced age, debilitated patients, previous empyema in the contralateral side with excessive decrease of the vital capacity preclude any major operative procedure; although occasionally the contralateral lesion may be only a small apical cavity.

Although it is true that with the present perfected technic and broadened indications of thoracoplasty the number of patients unfit for this operation becomes progressively smaller, none the less there are still patients who cannot stand any kind of major surgery. Such was the case of a white girl of sixteen years, who presented, besides bilateral apical cavities, luetic infection (Wassermann plus 4) and a marked arsenic neuritis following intensive arsenic treatment. In this patient apicolysis with plombe was performed on the right side and pneumothorax on the left. An excellent result was obtained in this case. The same technic was carried out on a man of thirty-five years, with a bilateral apical lesion. Marked emphysema complicated that case, prohibiting thoracoplasty. Apicolysis with plombe was performed on the right apex; the left lung was collapsed with pneumothorax which was completed with closed pneumonolysis. Later, as the plombe did not succeed in obliterating the right cavity, it was removed and thoracoplasty was performed. This patient has markedly improved but still has occasionally positive sputum (Gaffky 1-2).

In another case, a man of twenty-eight years, with an advanced bilateral lesion, located in the upper third of both lungs, with advanced gastrointestinal tuberculosis, low hemoglobin, and bad general condition prohibiting thoracoplasty, bilateral apicolysis with plombe was performed and he showed marked improvement both clinically and bacteriologically. He died, however, nine months later, following ulceration through the left apex from the plombe. (See cases.)

To sum up, no bilateral case should be considered hopeless as long as one-fourth of the lungs is free from disease. When pneumothorax is impossible, thoracoplasty in several stages should be performed, bilateral or combined with pneumothorax on the other side. Apicolysis with plombe, permanent or temporary, is only exceptionally indicated. Phrenic nerve interruption, temporary or permanent, should be avoided except in cases of basal lesions complicated by lesions of the upper lobe of the other lung. Bilateral phrenic operations are to be condemned.

TECHNIC

The procedures of pneumothorax, Jacobaeus, thoracoplasty, apicolysis with plombe, phrenic nerve crushing or exeresis are being performed, in a general way, with the technic usually followed in our institutions for unilateral cases. A detailed description of our technic has lately

been given elsewhere.[1, 15] There are, however, a number of details which should be pointed out.

(a) *Pneumothorax.*—The main objective of pneumothorax in bilateral cases should be the rapid production of a good selective collapse. Therefore, when adhesions are present, no time should be lost in trying positive pressures. The adhesions should be sectioned as soon as possible. Even if they appear in the roentgenograms as impossible to be handled by closed pneumonolysis, a thoracoscopic exploration should be done in all cases in which there is a sufficient amount of air in the chest to allow the introduction of the thoracoscope without danger of injuring the lung.

Complete section of the adhesions in apical lesions renders possible the production of a strictly selective collapse, limited to the upper lobe, while the healthy lower lobe is allowed to reexpand. Thus the vital capacity is increased and contralateral pneumothorax or thoracoplasty can be attempted with less respiratory discomfort for the patient.

(b) *Pneumonolysis.*—Our attempts of open pneumonolysis in the bilateral cases have been discouraging. We believe that closed pneumonolysis is a better and safer procedure. The thoracoscope gives a greater radius of action and better visibility in the chest than an incision made in an intercostal space, unless we use very long incisions. The question which we are not yet prepared to answer is whether in advanced bilateral cases we should attempt to liberate the lung from the parietal pleura by the method described by Toreck when pneumothorax could not be induced by the usual method. Recently the procedure was tried in a woman thirty-two years old, presenting advanced apical lesion with no untoward postoperative effects. The case is too recent to allow any conclusions.

The procedure of Jacobaeus has been a great help in our work. We can even say that it has been this procedure which has rendered it possible. The study of our statistics will show the surprising results that we have obtained by enlarging the scope and the indications of this procedure. Thus far it was generally considered that only thin and long adhesions should be cut; short and thick adhesions, in which the lung parenchyma would be included, should be carefully avoided in order to prevent possible injury to the lung, formation of bronchial fistulas, or production of hemorrhage. Such caution in technic cannot be applied to bilateral cases. In these cases all adhesions should be cut; even when the lung is directly attached to the parietal pleura we should endeavor to dissect it free. Only thus has it become possible successfully to manage bilateral cases.

A priori such a radical technic should expose the patient to dangerous complications. This is not the case if we follow the technic we have developed and upon which only a few words can be said in this paper.

For this purpose only instruments with cautery, separate from and independent of the thoracoscope, can give the necessary freedom of movements; only direct thoracoscopes presenting no or very slight deviation of the optical ray should be used. Furthermore, the electrocautery knife gently heated to a dull red is to be preferred to diathermic electrodes because the first controls bleeding better and does not expose to the danger of faradic effect upon the vagus nerve which may cause arrest of the heart and death. We have had the misfortune to have such a catastrophe.

Broad bands or direct attachments of the lung to the parietal pleura are attacked as follows: an incision of the pleural layer covering the adhesion alone is made at the junction of the visceral and parietal pleurae. Then the current is turned off, and with the cold electric knife acting as a blunt dissector we endeavor to discover a line of cleavage between the lung and the chest wall. Most of the time such a plane is found. In a number of cases the operation can be completed in one stage. More often, however, several stages are necessary for complete dissection of such adhesions. Between stages, tension due to the weight of the lung and to pneumothorax gradually causes the elongation of the adhesion the resistance of which has been weakened by the previous partial section. In four cases, four stages were necessary on one side and two on the other in order to produce complete bilateral selective apical collapse and persistently negative sputum (see cases).

In order to render this technic possible, radical changes were made in the Jacobaeus-Kremmer apparatus; they will be described elsewhere.

This technic is certainly dangerous in untrained hands. It requires long training, but like most technics it can be mastered. At any rate, as things stand today this technic is, we believe, the most important single factor concerned in the management of bilateral cases.

(c) *Thoracoplasty.*—The usual technic of thoracoplasty requires only minor changes to be adapted to bilateral cases. No more than three ribs should be resected at a time. In case of marked tendency to paradoxical respiration it is advisable to resect only a short segment of the third rib. Conversely the apex should always be stripped from the vertebrae and the sternum according to the technic of Carl Semb.

The second stage is more dangerous than the first stage because a larger segment of the lung is affected by the resection of ribs than at the first stage. Therefore it is often necessary to remove only two ribs. With these precautions thoracoplasty is no more dangerous in bilateral than in unilateral cases.

The same care should be taken to avoid massive decrease of the vital capacity and sudden reduction of the respiratory field in bilateral thoracoplasties. The greatest number of ribs resected by us so far is 7 on one side and 5 on the other side. In four of our seven cases the vital capacity dropped from 1,500 to 900 c.c. These patients, however,

are not bedridden; they are all able to move about, and one of them (A. R.), in whom 3 ribs were resected on the left side and 6 on the right side, has been performing a full day's work for the past ten months.

The majority of bilateral cases expectorate before operation from 1 to 5 ounces of sputum; it is therefore wise to use during operation the intratracheal catheter of Coryllos and administer the anesthetic through it. Bronchial exudate should be aspirated before, during, and after operation in order to avoid retention of the exudate and bronchial obstructions.

(d) *Apicolysis With Plombe, and Phrenic Nerve Interruption.*—The technic of these operations is exactly the same in bilateral as in unilateral cases.

COMPLICATIONS

Unfortunately the surgical treatment of bilateral pulmonary tuberculosis is fraught with numerous and dangerous postoperative complications. The most important of these complications which have occurred in our cases are:

1. Massive spread of the disease to the lower lobe or to the contralateral lung. This complication was frequent in our first bilateral cases, following thoracoplasty, when we did not endeavor to check the increased activity of the contralateral lung by previous selective collapse of the contralateral lung. Since this measure has been carried out systematically, the number and severity of these complications have considerably decreased.

2. Transient massive infiltration (probably allergic in nature) of the contralateral lung: This peculiar complication has occurred in two of our cases in which the contralateral lung was partially collapsed by pneumothorax. We shall report here briefly one of these cases.

J. Z., white, female, nineteen years, bilateral apical lesions with pneumothorax. A first-stage thoracoplasty (3 ribs) was performed on the right side. Postoperative course uneventful. One month later a second-stage thoracoplasty was carried out (3 ribs). The immediate postoperative course was smooth. On the eighth day the patient became greatly dyspneic and cyanotic. She was gasping for air. Temperature rose to 103°. Roentgenograms showed a diffuse opacity, mottled in aspect, occupying the whole left lung. The patient was placed in oxygen tent but without any relief. It was apparent that the immediate danger of asphyxia was due to a considerable decrease of the respiratory field. This anoxemia acted upon the respiratory center starting a vicious circle of shallow rapid breathing, excessive elimination of CO_2, peripheral blood stasis and excessive drop in blood pressure. It was thought that if we could assure a regular mechanical respiration independent of the action of the respiratory center, we might succeed in breaking this vicious circle. In other words, the problem was whether by replacing her respiratory center with a mechanical respiratory center situated outside of her organism and not influenced by the humoral changes of the blood of the patient we might be able to weather the storm. This patient was placed in Drinker's respirator regulated at 30 respirations per minute, and at the same time O_2–CO_2 was administered. The condition of the

patient showed marked improvement as long as she remained in the respirator. After sixty hours she was taken out. Six hours later she presented marked dyspnea. We again placed her in the respirator, where she remained for nine days. Only short interruptions of the apparatus were allowed, for the usual care and x-raying of the patient. The left chest cleared up gradually and dyspnea disappeared. The patient was taken out and made a normal recovery. The most surprising phenomenon was that on the twenty-fifth postoperative day the sputum became negative and has remained so for three months. Actually it is negative.

The second case is that of a white male, forty years of age, who had presented similar symptoms. He was kept in the respirator for four days. He made an excellent recovery and was discharged from the hospital with persistently negative sputum.

3. *Spontaneous Pneumothorax on the Contralateral Side.*—From the start we were afraid of the catastrophes that might occur in submitting to thoracoplasty patients who had pneumothorax on the other side. It seemed to us that it would be quite impossible for a patient, whose breathing area was already greatly decreased, to overcome an additional and sudden reduction caused by the production of spontaneous pneumothorax.

This complication did occur 22 times in 16 patients. Four of these patients died, and 12 recovered. In 3 of the 4 who died, pneumothorax occurred during or immediately after the operation. Only once it developed on the seventh postoperative day following a first-stage thoracoplasty. This patient recovered.

Two patients had spontaneous pneumothorax following each of their three operative stages, and two had two.

The diagnosis of this complication is easy when we look for it. In two of our cases, however, it was not made in time, and the patients died. Further experience makes us believe that these patients could have been saved. The onset is occasionally marked by severe pain and sudden dyspnea and cyanosis. The outstanding symptoms are bulging and immobility of the affected hemithorax, displacement of the heart and trachea to the other side, absence of breath sounds and, occasionally, amphoric breathing. The only real pathognomonic symptom, however, is a rise of the intrapleural pressure. Therefore, these patients should be supervised very closely and an apparatus for easy detection of rise of intrapleural pressure should be in readiness. This apparatus can be easily assembled. It consists of a bottle half filled with water; through its stopcock pass 2 glass tubes, one of which is connected with a rubber tube, 25 cm. long, attached to a pneumothorax needle. When spontaneous pneumothorax is suspected, the needle is introduced into the pleural cavity, while the end of the glass tube is placed at only $\frac{1}{16}$ in. under surface of the water. Bubbling of air through the water in the bottle confirms the diagnosis of spontaneous pneumothorax.

The same apparatus serves for continuous deflation, which is the treatment of choice of this complication. For this purpose the needle of the pneumothorax is passed through a large cork stopper so that only the length of the needle necessary to reach the pleural space protrudes through the cork. Thus injury of the lung is prevented. The needle is introduced into the chest and made secure by fixing the cork to the chest wall with bands of adhesive. It is left in place as long as bubbling of air continues in the bottle. Gradually the bubbling ceases and the water rises in the tube, indicating closure of the pneumonopleural communication and negative intrapleural pressure. The duration of deflation may vary from a few hours to several days. Occasionally the volume of air escaping into the pleura is so large that it cannot be handled by the needle. In these cases the latter should be replaced by a cannula or even by a rubber tube introduced into the chest and connected with the same apparatus.

The prognosis of this complication, when it is diagnosed and treated immediately, is surprisingly benign. In fact, we deal here with spontaneous pneumothorax presenting all the characteristics of idiopathic nontuberculous pneumothorax and none of true tuberculous pneumothorax. The air is absorbed within a few hours after the closure of the pulmonary fistula and no fluid develops.

The pathogenesis of this peculiar complication was puzzling. We could not understand how a spontaneous pneumothorax having all the nontuberculous characteristics could develop in a tuberculous patient. The explanation was found by direct thoracoscopic examination which disclosed the presence of superficial translucent emphysematous blebs on the surface of the lung. These findings made clear the pathogenesis of this complication.

Detailed study of this interesting complication will be presented later.

4. *Massive Atelectasis. Intratracheal Aspiration.*—This complication is the most dangerous of all accidents which may occur during the twenty-four or seventy-two hours following thoracoplasty in bilateral cases. The onset may be extremely sudden and may cause death within a few minutes. The symptoms are the same as those in any postoperative massive atelectasis. They are more dramatic in bilateral tuberculous patients because of the already markedly reduced respiratory field and chronic anoxemia. Noninformed observers are confused and stand by the patient helpless, thinking of embolism or cardiac or respiratory failure. In fact, these are asphyxial deaths due to acute anoxemia caused by obstruction of bronchial exudate of large bronchi. The therapeutic indications are clear, and treatment should be applied without any loss of time. Seconds count in these cases. Treatment consists in the introduction of the tracheal catheter and suction of the exudate. In very marked dyspnea no time should be lost in trying

the manipulations usually applied in atelectasis following abdominal operations, with the hope that the patient may cough out the obstructing mucus. Post-thoracoplasty patients are, as a rule, chronically anoxemic, and the slightest acute increase of anoxemia may cause rapid death.

This complication was produced in 4 of our cases. All of them were saved by immediate bronchial aspiration. The history of one of the cases gives a good picture of this complication.

C. P., thirty-two years of age, colored female. Cavities in left lung. Suspicious lesions in the right apex. Pneumothorax could not be induced on left side. Diagnostic pneumothorax on right side showed no cavity and was discontinued.

First stage thoracoplasty Sept. 24, 1935. Twenty hours after operation patient suddenly became extremely dyspneic and cyanotic; pulse hardly palpable. Patient became cold and clammy and lapsed into coma. No breath sounds over right lung.

A few minutes after examination, at her bed and without any anesthesia, intratracheal catheter was introduced and 20 c.c. of thick greenish yellow sputum was aspirated from the right common bronchus. As aspiration progressed, bubbling râles developed in the right lung; at the completion of aspiration breath sounds could be heard all over the right lung. Twenty minutes after aspiration patient was again conscious and coughing. She did not remember anything that had happened. Uneventful recovery.

This spectacular recovery of a patient who for all intents and purposes was already dead showed the importance of adequate postoperative supervision and care of these patients. As a rule, these patients do not die during the operation. The real danger which begins with the end of the operation is one: anoxemia. Only a well-trained medical and nursing staff, good teamwork, and promptness of action based upon knowledge of the pathologic physiology of these complications can produce such a brilliant recovery as that described above.

5. *Pleural Effusion. Tuberculous Empyema.*—Mixed infection empyema, following pneumonolysis. Pleural effusion occurs in about 60 per cent of the cases following closed pneumonolysis. As a rule, this fluid remains sterile and is gradually absorbed. In about 10 per cent of the cases it becomes purulent and presents the clinical characteristics of pure tuberculous empyema. In 3 cases, however, tuberculous empyema was present before pneumonolysis became secondarily infected. Only in one case purulent mixed infection developed due to Jacobaeus' operation. In the 3 cases a thick plastic exudate covering both visceral and parietal pleurae was found at thoracoscopy.

It is surprising that aggressive pneumonolysis can be carried out without any more complications. Repeatedly at the line of the section of the adhesions small caseated tubercles were opened without any untoward effects. When one considers that pneumonolysis is often prolonged over one hour, one really wonders at the resistance of the pleura to infection and mechanical injury. It is true, on the other hand, that heat can be better supported in the chest than in any other

cavity of the body; the considerable amount of blood circulating through the lungs represents an ideal cooling system.

Conversely the development of fluid in the chest was followed in a number of cases by unexpectedly favorable results. Thus, in 28 per

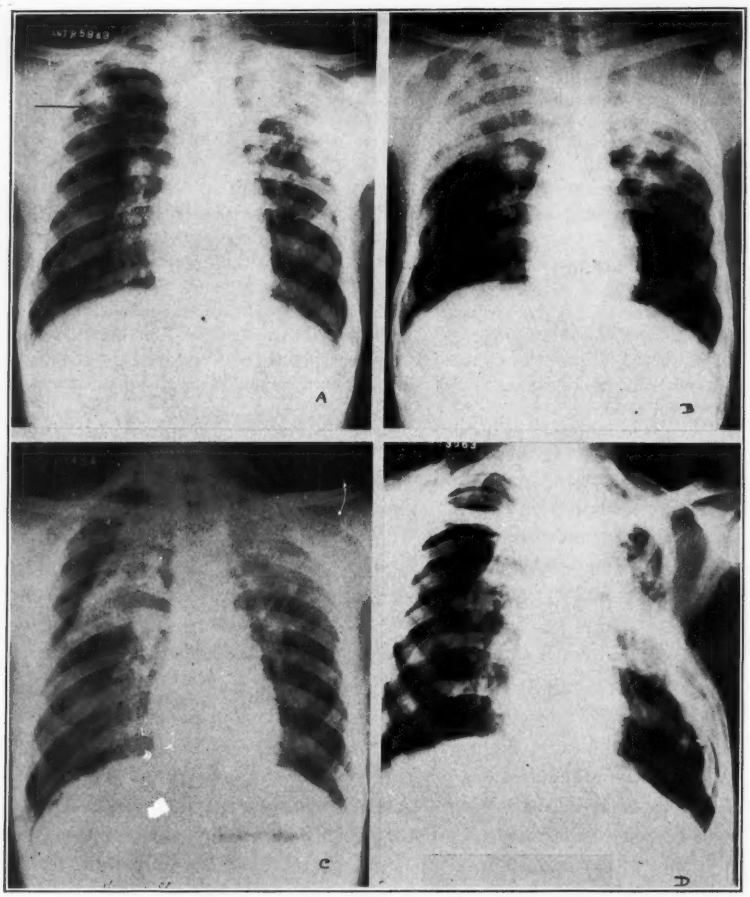

Fig. 1.—*A*, Multiple cavities in left apex. Small cavity in right side, July 15, 1933. Pneumothorax was induced in right side.

B, Aug. 15, 1934, upper stage left thoracoplasty followed by extensive spread in right upper lobe (July 31, 1934).

C, Oct. 29, 1934, pneumothorax was induced in the right side.

D, Sept. 24, 1935, second stage thoracoplasty was performed on left side (Nov. 13, 1934). Right upper lobe was maintained collapsed. Later this patient requested operation on left side. He died of extension of the disease Oct. 12, 1935.

cent of the patients on whom no section of adhesions could be done because of their size, the sputum became negative. This result could not be attributed to any other cause—outside of a spontaneous closure,

not related to the thoracoscopy—than the thickening of the visceral pleura and more marked and more steady collapse of the affected lung caused by the effusion.

CASES ILLUSTRATING THE MANAGEMENT OF BILATERAL PULMONARY TUBERCULOSIS

A small number of cases are presented here corresponding to the five groups studied in this paper.

Group 1. Cavities in one apex; suspicious lesion in the other.

The first case shows the aggravation of a small lesion present in one lung following thoracoplasty upon the other lung.

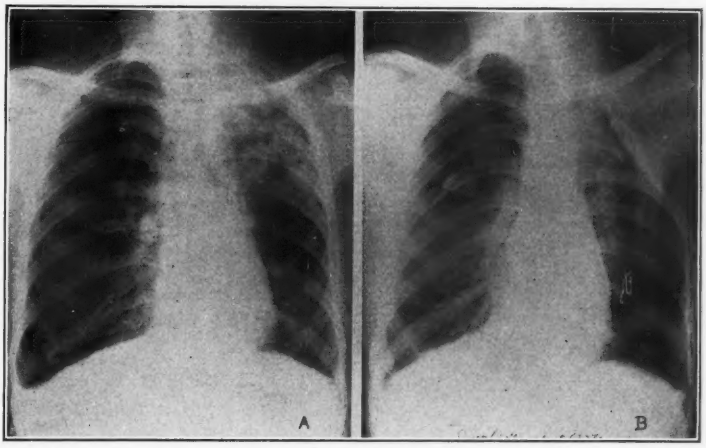

Fig. 2.—*A*, Multiple cavities in upper left lobe. Small cavity in right apex (arrow). Pneumothorax in right lung produced a good collapse of the upper lobe (Jan. 15, 1934).

B, Sept. 4, 1935, two-stage (6 ribs) upper thoracoplasty on left side with closure of cavities. Selective pneumothorax was maintained in right side. Sputum persistently negative.

CASE 1.—V. G., white male, eighteen years, admitted to Sea View Hospital July 15, 1933. Extensive caseous pneumonic lesions with multiple cavities in upper left lung. Small lesion in right apex (Fig. 1 A).

Patient was submitted to thoracoplasty without previous pneumothorax in left chest. Left pneumothorax had been tried without success on Nov. 1, 1934. First-stage (left) thoracoplasty (3 ribs) on right side (July 31, 1934). Extensive spread developed in the right apex soon after left thoracoplasty (Fig. 1 B). Pneumothorax was induced on right side with production of a good selective apical collapse (Fig. 1 C). Second stage thoracoplasty (3 ribs) was performed on Nov. 13, 1934 (Fig. 1 D). Condition improved surprisingly but sputum remained positive. Patient died a year later (Nov. 12, 1935) with extension of the disease.

This case shows the danger of neglecting a lesion in the contralateral lung, apparently unimportant. It is possible that if pneumothorax had been successfully induced in the right apex this lesion could have been controlled during the post-operative period by selective pneumothorax. Case 2 illustrates this point.

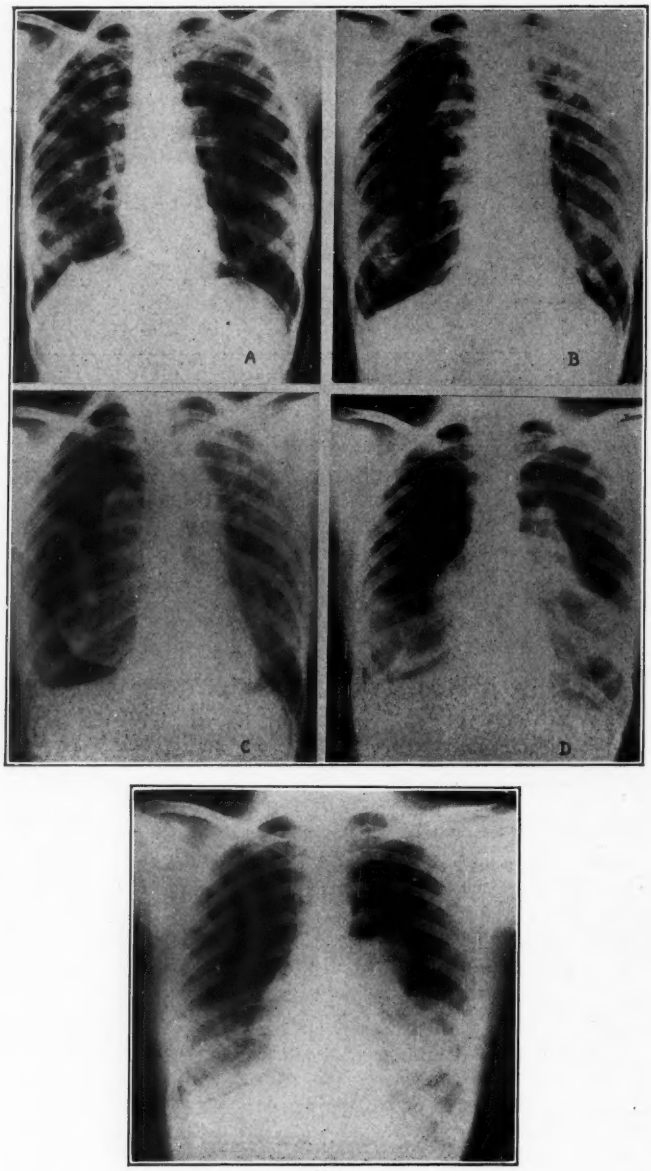

Fig. 3.—*A*, April 18, 1933, apical cavities in left lung. Multiple cavities in right lung (upper).

B, Oct. 2, 1934, poor collapse of right lung by pneumothorax because of adhesions.

C, Jan. 8, 1935, gradual section of adhesions in several stages. A few adhesions still hold the apex. Cavities cannot be seen in right apex.

D, Nov. 12, 1935, the right upper lobe is completely collapsed; right lower lobe was allowed to reexpand.

E, Nov. 29, 1934, the adhesions of the upper left lung were sectioned in two stages, and complete selective collapse of this lobe was obtained. Sputum negative since December, 1935.

CASE 2.—J. D., white male, twenty-one years. Onset June 3, 1933. Admitted to Sea View Hospital Jan. 9, 1934. Multiple cavitations in left upper lung. Small cavities in upper right lung. Pneumothorax was induced in right lung Jan. 15, 1934 (Fig. 2 A). It was maintained throughout the operation and postoperative period of two stages of thoracoplasty (6 ribs) performed on left side (April 16 and May 5, 1935). Sputum became negative four weeks after second stage. The

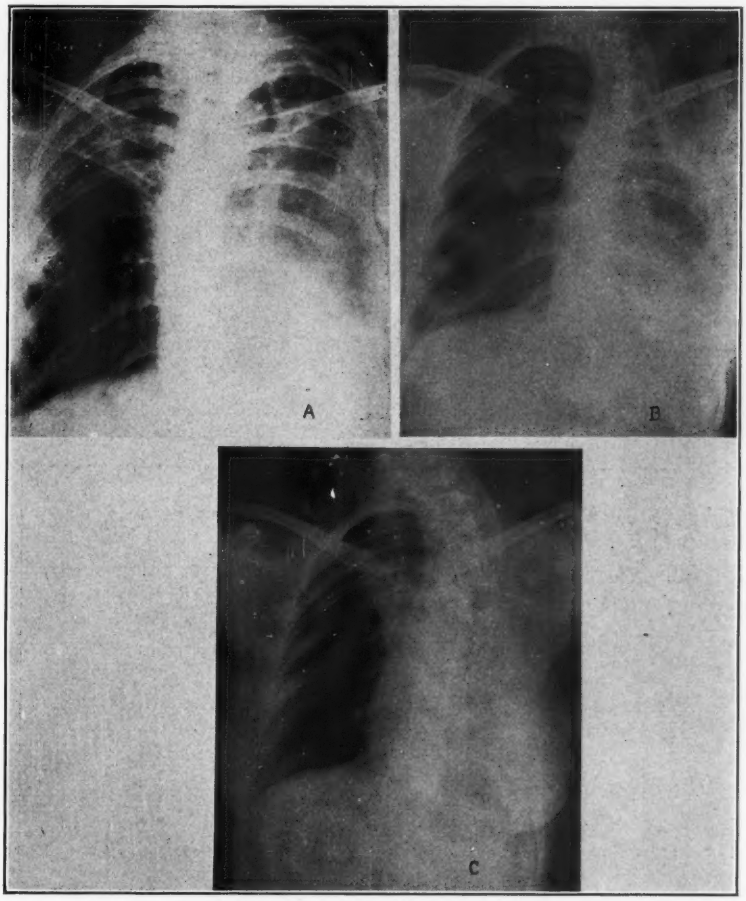

Fig. 4.—*A*, Nov. 14, 1932, large cavity in left apex. Multiple cavities in right upper lobe.

B, Oct. 2, 1933, selective pneumothorax of the upper right lung was obtained. Two-stage thoracoplasty (6 ribs) was performed on the other side. Sputum persisted positive.

C, March 6, 1934, revision of previous thoracoplasty and resection of four more ribs produced complete collapse of left chest. Right upper lobe was allowed to reexpand. Sputum became negative December, 1934, and has remained so since.

right lower lobe was allowed to reexpand completely, while a selective apical collapse was maintained (Fig. 2 B). Patient has been persistently negative for the past six months.

Group 2. Cavities are located in the apices on both sides.

Five cases will be presented illustrating five different ways of management of these cases.

(a) Bilateral pneumothorax completed by pneumonolysis on both sides.

(b) Pneumothorax on one side and thoracoplasty on the other side.

(c) Apicolysis with plombe on one side; pneumothorax completed by closed pneumonolysis on the other side. Plombe removed. Thoracoplasty two stages.

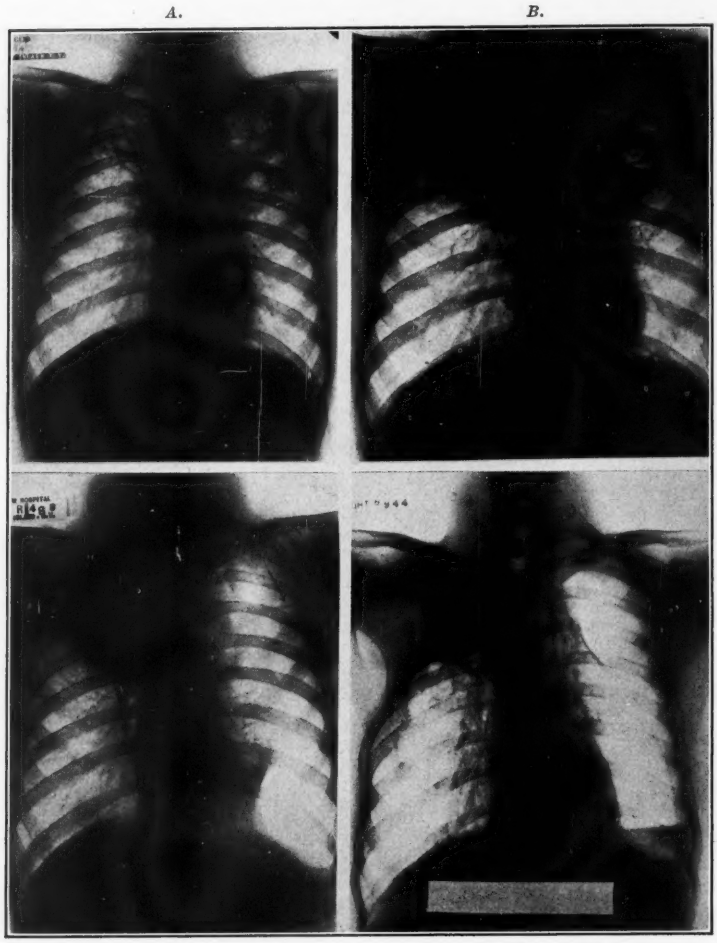

Fig. 5.—*A*, April 1, 1932, extensive bilateral multiple lesions, especially in left upper lobe.

B, June 10, 1932, apicolysis in the right apex with plombe.

C, Feb. 19, 1933, pneumothorax was induced in left side; strong adhesions were present (arrow) preventing closure of cavities.

D, March 25, 1933, adhesions were sectioned (Feb. 23, 1933) and an excellent collapse of the left lung was obtained. Because of persistence of positive sputum, plombe was removed from right apex and a two-stage thoracoplasty (6 ribs) was performed. Sputum negative with occasional Gaffkey 1-2 on concentration.

(d) Bilateral apicolysis with plombe.
(e) Bilateral thoracoplasty.
Bilateral pneumothorax completed by staged pneumonolysis on both sides.

CASE 3.—A. N., white female, twenty-seven years. Onset 1928. She was discharged from another institution in 1929 as an arrested case. Admitted to Sea View Hospital on Sept. 15, 1934. This case belongs rather to the third group because of the extension on the right side (Fig. 3 A).

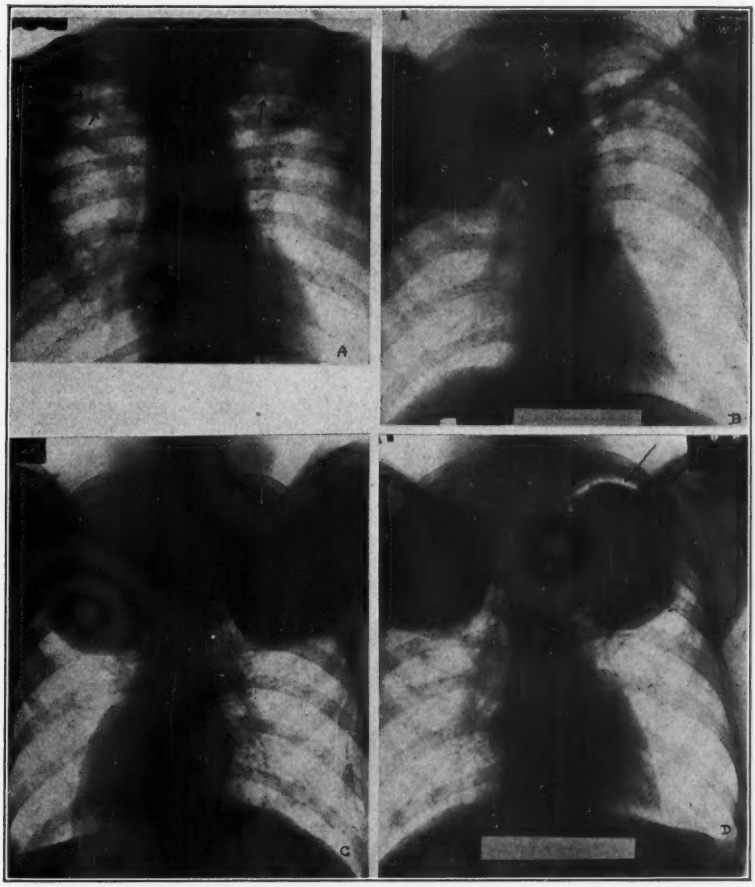

Fig. 6.—*A*, Jan. 8, 1932, multiple bilateral cavities.
B, Jan. 19, 1932, eighteen days after apicolysis with plombe in right side Feb. 1, 1932. Sputum positive.
C, March 26, 1932, a month after apicolysis with plombe in left side Feb. 28, 1932.
D, July 7, 1933, sixteen months after apicolysis. Perforation of left apex (air above paste in left side, shown by arrow). Rapid spread of the disease and death.

Pneumothorax was induced on the right side producing a poor collapse of the lung because of numerous adhesions. At the first thoracoscopic examination the case appeared hopeless. Large and short adhesions were present, and the lung was stuck posteriorly to the parietal pleura (Fig. 3 B). Three successive attempts for pneumonolysis were made (Nov. 10, 1934, Feb. 7 and June 10, 1935).

Each time as many adhesions were cut as the condition of the patient allowed. Large adhesions were only partially sectioned at the first stage. The intrapleural pressure was increased so that, at the second stage, section of the elongated adhesions was easier and the lung was dissected free from the chest wall; only a group of apical adhesions was left (Fig. 3 C), which was sectioned at the third stage. Thus

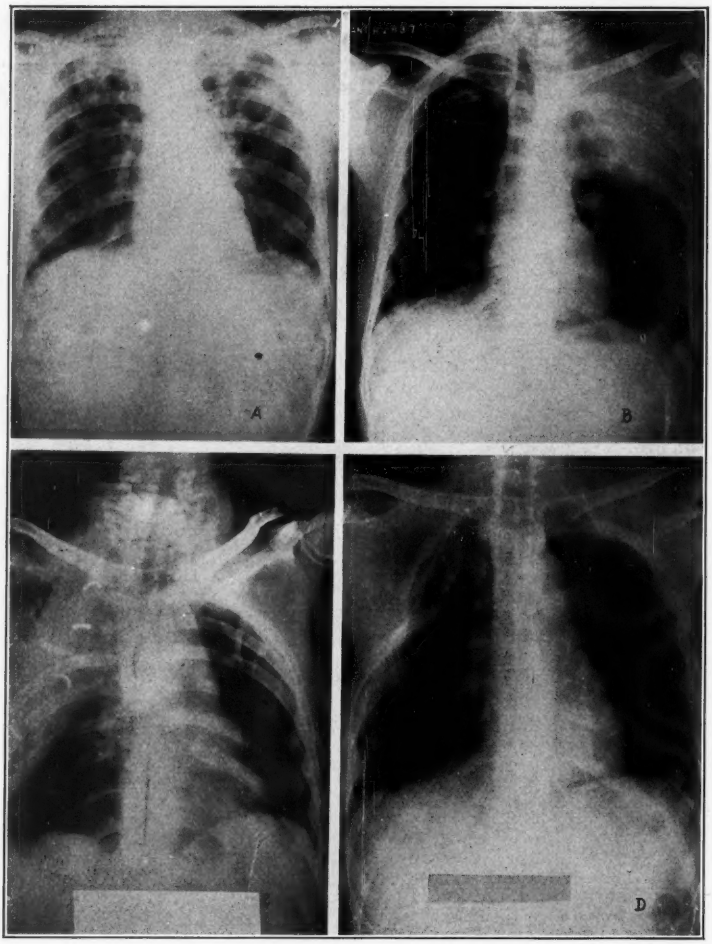

Fig. 7.—*A*, Sept. 22, 1933, multiple bilateral cavities, especially in left upper lobe. *B*, March 12, 1934, pneumothorax could not be induced in either side. First stage thoracoplasty (3 ribs) was performed in the left side (March 1, 1934). *C*, May 31, 1934, postoperative radiograms following second stage thoracoplasty in right side (6 ribs) (April 5 and May 26, 1934). *D*, Aug. 8, 1934, cavities in both upper lobes are closed. Sputum persistently negative on concentration. Patient discharged.

a selective collapse of the whole right upper lobe was obtained. The right lower lobe was allowed to reexpand, and pneumothorax was induced on the left side (Fig. 3 D). This was also incomplete because of adhesions. The latter were sectioned in

two stages (Sept. 9, 1934, and Nov. 14, 1935), and selective collapse of the whole left lung was obtained (Fig. 3 E). A week after the last pneumonolysis the sputum of the patient, which previously was highly positive (Gaffky 8-10), became negative and it remained so since.

Pneumothorax on one side and thoracoplasty on the other.

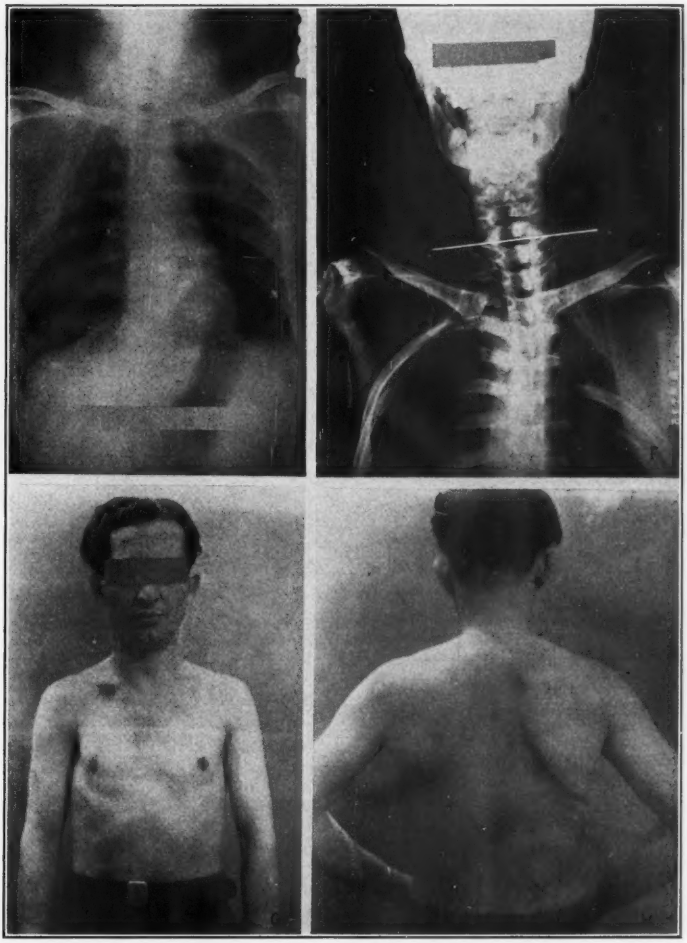

Fig. 7.—*E-H*, same case as shown in Fig. 7 *A-D*.
E, April 19, 1935, eight months later, in the follow-up clinic. Patient working.
F, Aspect of the skeleton following bilateral thoracoplasty, giraffe neck.
G, and *H*, Photographs of the patient. There is neither deformity nor functional impairment.

CASE 4.—E. F., white female, twenty-four years. Onset October, 1930. Admitted to Sea View Hospital Nov. 11, 1932, with bilateral lesion. Large cavity in upper left lobe. Multiple cavities in right apex with a tendency to retraction (Fig. 4 A).

Pneumothorax was induced on the right side producing a satisfactory selective collapse. Two-stage thoracoplasty was performed upon the left lung. No aggrava-

tion occurred in the lesion of the right apex (Fig. 4 B) but sputum remained positive. Reoperation and completion of right thoracoplasty were done, and 4 more ribs were resected (Fig. 4 C). Pneumothorax was maintained on right for eighteen months. The right lung was then cautiously allowed to reexpand (Fig. 4 C); the sputum continued positive for four months after the completion of thoracoplasty (Gaffky 4, in

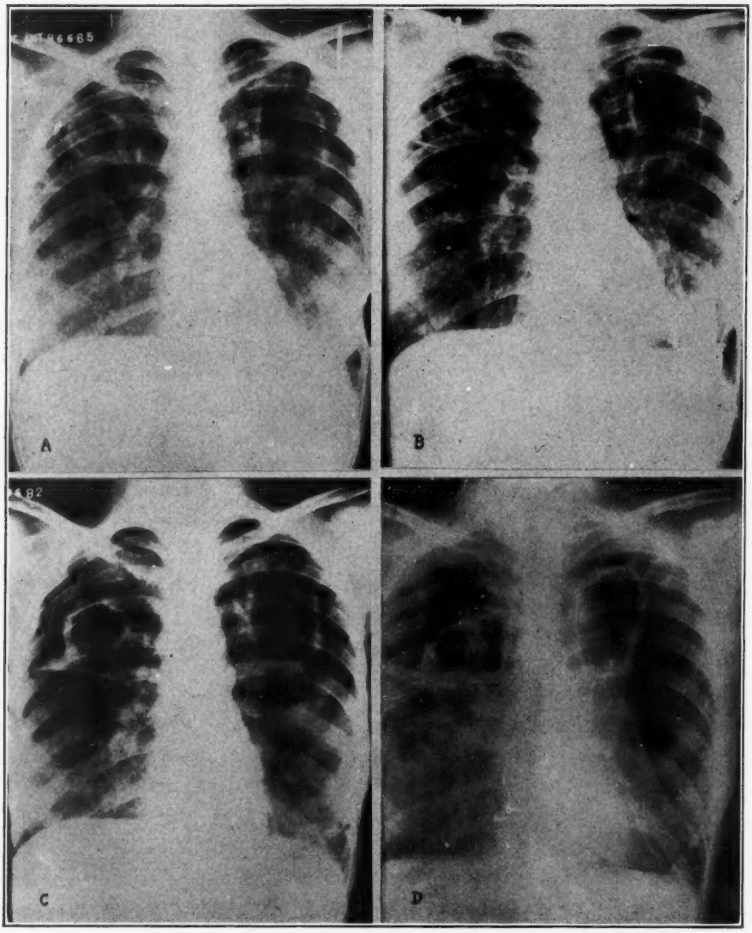

Fig. 8.—*A*, June 25, 1934, far-advanced ulcerative tuberculosis with multiple disseminated cavities in both lungs. Attempt at pneumothorax was made in the left side. (Posteroanterior.)

B, Same, anteroposterior.

C, Aug. 18, 1934, collapse of the right lung was unsatisfactory. Note that both cavities have increased in size under pneumothorax.

D, March 23, 1935, repeated attempts for section of the adhesions were made on both sides, considerably improving the collapse.

concentrated sputum). It became persistently negative at that time, and the patient was discharged. She has been seen regularly at the follow-up clinic since. Her condition has remained excellent.

Apicolysis with plombe on one side replaced later by thoracoplasty; pneumothorax completed by closed pneumonolysis on the other side.

CASE 5.—E. S., white male, twenty-six years. Onset 1930. He had been successively in Lenox Hill, Bellevue, and Riverside Hospitals in New York City. Admitted to Sea View Hospital on March 19, 1932.

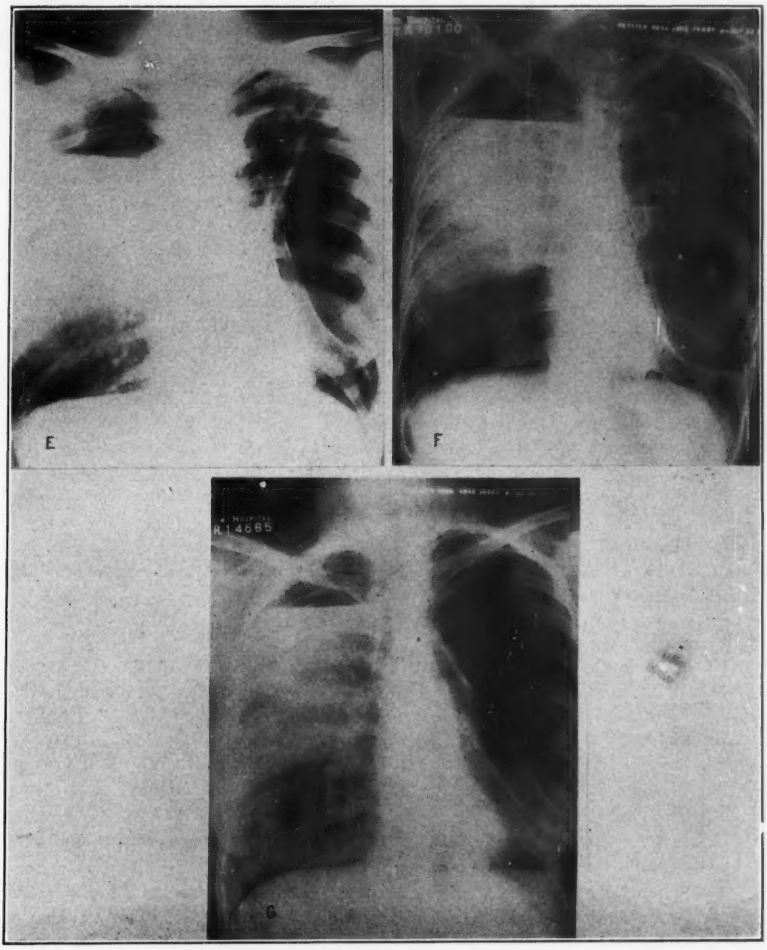

Fig. 8.—*E-G*, same case as shown in Fig. 8 *A-D*.

E, May 24, 1935, right apex was completely collapsed. The lower lobe is completely expanded. A slight amount of serous fluid is collected above the adherent lower lobe. Left lung is still adherent.

F, Postoperative films immediately after the last stage of pneumonolysis in the left side. All remaining adhesions were sectioned.

G, Perfect bilateral selective collapse of the upper lobes. Sputum became persistently negative. Eight weeks after Fig. 8 *F*.

Patient presented far-advanced bilateral caseous pneumonic lesions. Numerous large cavities were present in both upper lobes, especially on the left (Fig. 5 A). Pneumothorax could not be induced on right side. Patient was in bad general

condition, and we did not think that he could stand a thoracoplasty. Therefore, apicolysis with plombe was done on that side (June 10, 1932) (Fig. 5 B). Left pneumothorax was induced Feb. 10, 1933, which did not succeed in closing the cavities because of the presence of a strong adhesion (Fig. 5 C); this adhesion was sectioned by closed pneumonolysis a few days later (Feb. 23, 1933). A complete pneumothorax was obtained. The sputum, however, was persistently positive (Gaffky 3-6). Bronchoscopic smears taken separately from the right and left bronchi disclosed tubercle bacilli only in the right bronchus. Therefore, the apicolysis of the right apex was revised. The paste was removed and a two-stage thoracoplasty (6 ribs) was performed (March and April, 1934) (Fig. 5 D). Shortly after, the sputum became negative with only an occasional positive on concentration.

Bilateral apicolysis with plombe.

CASE 6.—P. McL., white male, twenty-nine years. Admitted to Metropolitan Hospital Nov. 11, 1931. Patient presented multiple large cavities in both upper lobes, with bronchogenic spread through both lungs (Fig. 6 A). Sputum Gaffky 8-10. Pneumothorax could not be induced on either side. Thoracoplasty was contraindicated because of his run-down condition.

Apicolysis on right side Feb. 1, 1932, under local anesthesia, by anterior approach (Fig. 6 B). Apicolysis on left side Feb. 28, 1932, by same technic (Fig. 6 C). Patient stood well both operations and showed a marked improvement. Expectoration decreased and weight increased 15 pounds by Sept. 30, 1932. Sputum remained positive (Gaffky 6-8). This improvement continued until August, 1933, when he complained of sudden pain and expectorated bloody sputum containing small pieces of paste. Radiogram showed air above the paste in left apex (Fig. 6 D). It was evident that the paste had perforated the left apex. Condition of patient became rapidly worse; massive bronchogenic spread in both lungs followed and patient died Nov. 15, 1933.

This case shows that bilateral apicolysis exposes the patient to this dreadful late complication of perforation of the lung. As this complication was followed by death in two of our cases, we abandoned this procedure.

Bilateral apical cavities—bilateral apical thoracoplasty.

CASE 7.—A. R., white male, twenty-four years. Onset May, 1932. He was in Bellevue Hospital for two months. Admitted to Sea View Hospital for eleven months. He signed out and was readmitted on Feb. 5, 1934. He presented multiple large cavities in both upper lobes. Lesion was more extensive in left lung (Fig. 7 A). Numerous attempts to induce pneumothorax on either side were unsuccessful. A first stage (3 rib) thoracoplasty was performed on the left side (March 1, 1934) (Fig. 3, 8), which produced closure of cavities.

A two-stage thoracoplasty (April 5 and May 26, 1934) was necessary to obliterate the cavities on the right side (Fig. 3 C).

Sputum became negative four weeks after the second stage on the right side. Patient was discharged and has been regularly followed since. Sputum has remained negative. No cavities have reappeared, as shown in Fig. 3 D and E. There is no deformity or impairment of function (Fig. 3 G and H) although 9 ribs have been resected (Fig. 3 F).

Groups 3, 4, 5. The cases responding to these groups are all far-advanced bilateral ulcerative cases. For sake of space we shall report only four cases; two of these cases were treated by bilateral pneumothorax, one by pneumothorax and thoracoplasty and the fourth by bilateral thoracoplasty. These cases will illustrate, we believe, the statement made in this paper that no bilateral case is hopeless unless and until every combination of collapse treatment has been tried and has failed.

CASE 8.—C. O'S., white female, twenty-five years. Admitted to Sea View Hospital. Giant cavities in right and left upper lobes; multiple cavitations disseminated in both lungs (Fig. 8 A).

Pneumothorax was attempted on the right side (July, 1934). After several attempts air was introduced around the upper lobe. However, no successful collapse

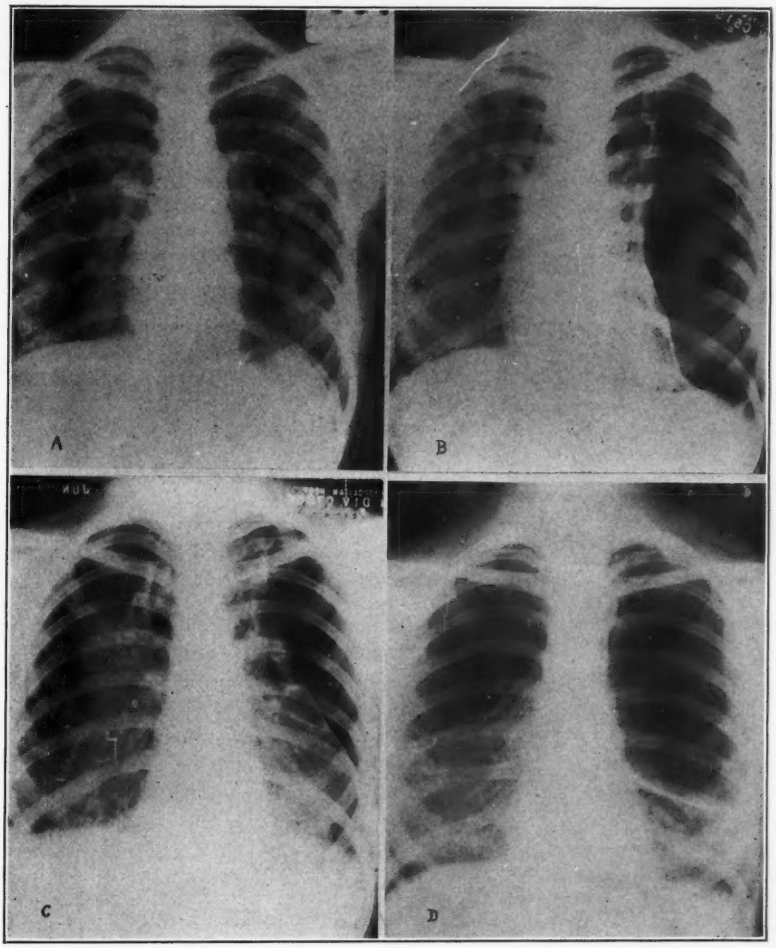

Fig. 9.—*A*, June 11, 1934, large cavities disseminated in both lungs.

B, Oct. 22, 1934, left pneumothorax with positive pressures. Cavities remained uncollapsed because of apical adhesions.

C, June 3, 1935, right pneumothorax did not yield any better results because of strong adhesions of the upper lung to the chest wall and to the mediastinum.

D, Nov. 21, 1935, adhesions were sectioned in several stages. Complete bilateral selective collapse of upper lobes. Cavities closed, sputum negative since October, 1935.

could be obtained because of numerous adhesions (Fig. 8 B). A month later (August, 1934) pneumothorax was induced on the left with similar unsatisfactory results as on the right side.

With great care to avoid injury to the lung, exploratory thoracoscopy was made on right side; the presence of a considerable number of short and thick adhesions, containing lung tissue, was shown. More exactly, the apex was in direct contact with and adherent to the chest wall (Fig. 8 A, B, C, D). Using the technic already described we were able in four repeated stages (March to May, 1935) to dissect the lung free from the chest wall, thus obtaining an excellent selective collapse of the

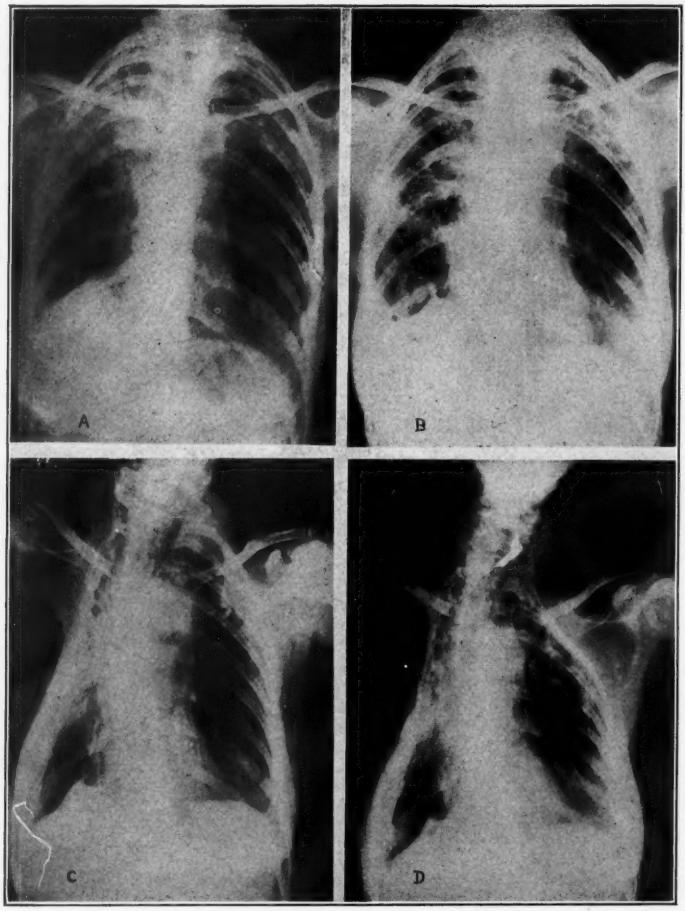

Fig. 10.—*A*, Feb. 25, 1933, extensive bilateral cavitations especially in the right.
B, Oct. 27, 1934, lesions have increased under bed rest.
C, March 25, 1935, two-stage thoracoplasty (7 ribs) was performed in the right side with good anatomic results (Nov. 20 and Dec. 11, 1934).
D, Oct. 10, 1935, upper stage thoracoplasty was performed on the right side May 4, 1935. Sputum became negative two weeks after this stage and remained so.

upper right lobe. A small amount of serous fluid developed above the interlobar fissure (Fig. 8 E, F, G, H) which was not removed. Since the right lower lobe was adherent, we were able to force the pneumothorax on the left side. The remarkable feature was that these repeated operative procedures and the marked decrease of respiratory field

were supported without any untoward effect. Pneumonolysis was carried on on left side; the adhesions were sectioned in two stages (June and July, 1935) and an excellent collapse of the left upper lobe was obtained (Fig. 8 F, H.). All cavities were closed; the sputum became negative in August, 1935, and has remained negative in concentration; repeated examinations of gastric lavage were negative. In comparing the first and the last radiograms of this patient, one has the right to wonder whether there are any limits to the operability and curability of pulmonary tuberculosis.

This is not an isolated and exceptional case. The following case shows the same results obtained by the same method.

CASE 9.—M. G., white female, twenty-two years. Admitted to Metropolitan Hospital. Patient had a far-advanced bilateral caseous pneumonic tuberculosis. Large cavities were disseminated in both lungs (Fig. 9 A).

Pneumothorax was induced on left side (Sept. 5, 1934). Notwithstanding positive pressures the cavities did not close because of strong apical adhesions (Fig. 9 B). Pneumothorax was then induced on the right side without succeeding in closing the

E.

Fig. 10.—*E*, Giraffe neck of patient shown in Fig. 10 *A-D*.

cavities because of apical and lateral adhesions (Fig. 9 C). Closed pneumonolysis was performed on both sides: three stages on right and two on left. All adhesions were cut and excellent selective collapse of both upper lobes was obtained (Fig. 9 D). Sputum became negative in October, 1935, and has remained as such, in concentrated sputa and gastric lavage. Gradually the lower lobes were allowed to reexpand, thus increasing the respiratory field. Patient is an ambulatory case. No dyspnea on moderate exertion. Vital capacity 1,100 c.c.

The following case shows the possibility of bilateral thoracoplasty upon advanced bilateral cases.

CASE 10.—Bilateral pulmonary ulcerative tuberculosis—bilateral thoracoplasty. M. M., white female, twenty-three years. Admitted to Sea View Hospital Dec. 29, 1930. Discharged as an arrested case October, 1929. Readmitted Oct. 27, 1934. Bilateral caseous pneumonic tuberculosis. Numerous cavities in both upper lobes; the lesion is extended in the whole right lung. This lesion has progressed considerably since 1933 (Fig. 10 A and B).

Pneumothorax could not be induced on either side.

Thoracoplasty performed upon right side in two stages (Nov. 20 and Dec. 11, 1934) (Fig. 10 C). Postoperative course following the second stage was very stormy. Patient was kept in Drinker's respirator for three days, and in oxygen tent for seven days. Vital capacity 950 c.c. Sputum remained positive (Gaffky 2-4).

Thoracoplasty on the left side with resection of 3 ribs was performed on May 4, 1935. Difficult postoperative course because of extreme dyspnea, ten days in oxygen tent. Patient rapidly improved. Present vital capacity of 900 c.c. Patient can do housework. Discharged with sputum persistently negative on concentration and negative gastric lavage.

STATISTICAL STUDY OF RESULTS

This study was based upon 238 cases, all presenting advanced bilateral tuberculous ulcerative lesions. All these cases were treated and followed during the last three years (1932-1935).

These patients can be subdivided into three classes.

Group 1.—One hundred and forty-four patients with unilateral or bilateral pneumothorax in whom we could not produce closure of the cavities because of the presence of adhesions. Pneumothorax in all these cases was completed on one or both sides by intrapleural closed pneumonolysis (Jacobaeus' procedure). In only 25 of the 144 cases, section of the adhesions could not be performed because of their size and extent. In this series we did not include cases of pneumothorax in which no other surgical procedure was performed.

Group 2.—Eighty-seven patients in whom pneumothorax could not be induced on one side and on whom thoracoplasty was performed instead.

Group 3.—Seven patients in whom pneumothorax could not be induced on either side and upon whom bilateral thoracoplasty was performed.

In tabulating our results we shall not use such expressions as cured, arrested, improved, unimproved, and worse. We shall divide our patients into only three classes. Negative sputum, positive sputum, and death.

Negative sputum means that the patients have presented persistently negative findings on concentrated sputums for over four months; furthermore that bacteriologic examination of at least three concentrated successive gastric lavages has shown no bacilli. No significance is attributed to clinical improvement and increase of weight; it is well known that such improvements are of no importance, as long as the sputum remains positive.

Plain smear examination is not sufficient for declaring a patient negative. Pinner has shown that 10,000 bacilli per c.c. are necessary to give positive results on plain smear; whereas only 500 are sufficient when the sputums are concentrated. On the other hand, we did not use cultures or inoculations because of the extreme sensitivity of these

methods; the presence of 1-5 bacilli in 1 c.c. of sputum suffices to give positive results. It is therefore problematic whether any patient with pulmonary tuberculosis can ever become bacilli free, bacteriologically speaking.

Group 1.—Unilateral or bilateral pneumothorax with unilateral or bilateral Jacobaeus' operation.

We deal here with patients who are considered as hopeless cases. In all these cases all attempts at efficient collapse with pneumothorax were unsuccessful. It is obvious that even the statistics of Barnes and Barnes with their mortality of 80 per cent within the first year can be considered as benign when applied to these cases. Therefore in judging the results obtained in these cases one should bear in mind that contrary to the unilateral cases, what really counts here is not so much the mortality but the number of patients rescued and rendered bacilli free.

Table I shows the total results obtained on these 144 cases.

TABLE I

BILATERAL CASES TREATED BY PNEUMOTHORAX, COMPLETED BY JACOBAEUS' OPERATION

HOSPITAL	TOTAL	NEGATIVE	POSITIVE	DEATHS
Sea View	92	25	55	12
Metropolitan	52	25	22	5
Total	144	50	77	17
Per cent		34.7%	53.5%	11.1%

Thirty-four and seven-tenths per cent of them became negative. However encouraging this result may appear, it is far from representing what can be done with these patients. In fact, in a number of them the cavities were closed in the collapsed lung following pneumonolysis, and the positive sputums were coming from the noncollapsed lung. This was repeatedly proved by taking separate specimens of sputum from the right and left common bronchi by means of the bronchoscope.

The real picture appears when we divide these cases into two subgroups; one, in which only unilateral pneumothorax completed by Jacobaeus was possible (Table II) and another in which bilateral pneumothorax with unilateral or bilateral Jacobaeus had been performed (Table III).

TABLE II

BILATERAL CASES. PNEUMOTHORAX. UNILATERAL JACOBAEUS

HOSPITAL	TOTAL	NEGATIVE	POSITIVE	DEATHS
Sea View	42	3	31	8
Metropolitan	30	10	18	2
Total	72	13	49	10
Per cent		18.0	68.0	14.0

In the 72 cases in which no pneumothorax could be induced on the other side, only 18 per cent became negative; 68 per cent remained

positive and 14 per cent died. The number of deaths must be considerably higher as a number of patients left the hospitals and could not be located.

On the contrary when a successful pneumothorax was obtained on one side and an incomplete pneumothorax on the other side could be completed by pneumonolysis, the results were different. Sputums became negative in 61 per cent of these cases instead of 18.8 per cent, and the mortality was nil.

TABLE III

BILATERAL CASES. BILATERAL PNEUMOTHORAX WITH UNILATERAL JACOBAEUS

HOSPITAL	TOTAL	NEGATIVE	POSITIVE	DEATHS
Sea View	24	14	10	0
Metropolitan	15	10	5	0
Total	39	24	15	0
Per cent		61.0%	39.0%	0%

The results were about the same when pneumothorax was incomplete on both sides but could be completed by successful pneumonolysis on both sides.

TABLE IV

BILATERAL CASES. BILATERAL PNEUMOTHORAX WITH BILATERAL JACOBAEUS

HOSPITAL	TOTAL	NEGATIVE	POSITIVE	DEATHS
Sea View	1	1	0	0
Metropolitan	7	4	2	1
Total	8	5	2	1
Per cent		62.5%	15.0%	12.5%

These results are instructive because they show the futility of maintaining for long periods incomplete pneumothoraces or even complete ones on one side only. On the other hand, they show the tremendous help that pneumonolysis performed early and skillfully can offer these desperate cases.

The counterproof of this fact is shown by the outcome of patients on whom pneumonolysis was attempted but was impossible because of the size and thickness of adhesions or the adherence of large portions of the lung to the chest wall.

TABLE V

UNILATERAL OR BILATERAL PNEUMOTHORAX. EXPLORATORY THORACOSCOPY WITHOUT SECTION OF ADHESIONS

SEA VIEW AND METROPOLITAN HOSPITALS				
Total	25	Negative	7	(28%)
		Positive	18	(72%)
		Deaths	4	(16%)

Of 25 patients 72 per cent remained positive and 16 per cent died. It appears reasonable to believe that the 28 per cent of good results obtained with these patients should be attributed either to the de-

velopment of pleural exudate following thoracoscopy or to spontaneous cures. At any rate the number of cases in which pneumonolysis was considered impossible is rapidly decreasing because of the modifications applied lately to the technic of this procedure as has already been mentioned.

Group 2.—Thoracoplasty with contralateral pneumothorax.

In 87 cases thoracoplasty was performed upon the side in which pneumothorax could not be induced. In 13 of these cases pneumothorax upon the other side was performed for diagnostic purposes only. In six, pneumothorax was induced after contralateral thoracoplasty, because of a spread in the noncollapsed lung. These patients were operated upon before our present procedure had been elaborated; in all these cases a small lesion could be seen in the radiograms previous to thoracoplasty. The results obtained by the combined pneumothorax and thoracoplasty procedure are shown in Table VI.

TABLE VI

BILATERAL CASES. PNEUMOTHORAX AND THORACOPLASTY

HOSPITAL	TOTAL	NEGATIVE	POSITIVE	DEATHS
Sea View	57	24	23	10
Metropolitan	30	18	6	6
Total	87	42	29	16
Per cent		48.2%	33.3%	18.4%

Forty-eight and two-tenths per cent of these patients became bacilli free, 33 per cent remained positive, and 18.4 per cent died. These results are less satisfactory than the ones obtained with bilateral pneumothorax and pneumonolysis. It is obvious, however, that we deal here with advanced cases which were submitted to a more strenuous surgical treatment. Therefore we have been rather surprised with the extraordinary results which we have had with the patients in the following Group 3: 56.1 per cent became negative, 2 remained positive, and none died.

Group 3.—Bilateral thoracoplasty.

BILATERAL THORACOPLASTY

Sea View Hospital 7; Negative 4 (56.1%); Positive 3 (43.9%); Deaths 0.

The only possible explanation is that these patients were in relatively good general condition. The onset of the disease varied between three and five years before operation, and they were free from myocardial damage and extrapulmonary foci. These results have encouraged us further to enlarge the operative indications for bilateral thoracoplastic collapse.

The results and statistical studies presented in this work show that the actually prevailing conceptions upon the indications for surgical

treatment of bilateral cases should be thoroughly revised. If we are correct in our conclusions, no bilateral case should be declared hopeless until all possible means of collapsing the diseased lungs have been tried. Abstention from active collapse treatment in these cases can by no means be labelled as conservative behavior. On the contrary, if our results have been correctly interpreted, it will soon be considered definitely negligent to adopt a *laissez faire* attitude of inactivity in such cases.

SUMMARY AND CONCLUSIONS

A study of the management of 238 cases of bilateral pulmonary ulcerative tuberculosis is presented. In all these cases pneumothorax could not be induced or it was unable to collapse the cavities, because of the presence of adhesions.

The additional application of two other procedures, namely, closed intrapleural pneumonolysis and thoracoplasty, was studied, and the results of this effort are reported. The results obtained show that 40 to 60 per cent of these cases, thus far considered as hopeless, can be rescued and their sputums rendered persistently bacilli free.

The indications for surgical treatment in these cases have been given. It is shown that the severe restrictions for surgical treatment commonly applied today upon these patients are not justified. Evidence is presented in favor of the conception that no case should be considered hopeless only because it is bilateral even when bilateral pneumothorax did not succeed in collapsing the cavities. The importance of a modified technic of closed pneumonolysis was stressed, and the efficiency of bilateral thoracoplasty in selected cases was insisted upon.

The surprisingly small amount of healthy pulmonary parenchyma necessary for continuation of moderately active life was a great aid in allowing us to collapse large areas of diseased lung successfully.

A statistical study of the results obtained is presented.

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DISCUSSION

DR. DEAN B. COLE.—I am impressed with the number of thoracoplasties shown by Dr. Coryllos on patients apparently well suited for pneumothorax. I should like to ask whether pneumothorax was attempted with each of these?

For the past two and one-half years we have tried pneumothorax in practically every patient before having a thoracoplasty performed. We have been agreeably surprised to find that in practically all these patients collapse will occur if pneumothorax is persisted in, and we believe the only way to determine whether or not pneumothorax is possible is by introduction of a needle. We have also found that we can close many cavities, which are previously thought impossible to close, by the use of pneumothorax combined with an apical thoracoplasty. We have been more successful with this procedure than with using the apical thoracoplasty without the pneumothorax. In such patients we do not let the lung reexpand prior to the thoracoplasty but introduce the needle at the time of operation so as to control the intrapleural pressure while the cavity is being closed surgically. We have not had a spontaneous pneumothorax in any such case.

We endeavor to close cavities wherever possible with our patients and find pneumothorax our most successful method. One of our patients who had pneumothorax combined with apical thoracoplasty was in bed only fifteen days following his thoracoplasty.

DR. G. G. ORNSTEIN.—The patients whom Dr. Coryllos has discussed are from two of the municipal hospitals in the city of New York. Because of the advanced disease in the cases referred to Sea View and Metropolitan Hospitals, thoracic surgery has been confined to a great extent to the bilateral cases of pulmonary tuberculosis. Our interest naturally had to be in the patients with bilateral disease. There is a uniform opinion now in regard to the management of the unilateral case. With the large number of advanced cases of bilateral pulmonary tuberculosis, new problems in thoracic surgery arose.

Our incentive came from the supposedly unilateral case with an apparently healed tuberculosis in the opposite lung. Reviewing some of the failures of thoracoplasty in this group, we found that the apparently quiescent lesion had spilled down the lung. We determined from then on to try to be certain in our determination of the disease in the good lung. The patient was sent to a special clinic in which the fluoroscopic examination was the important one. If there was any question of the presence of small cavities, a diagnostic pneumothorax was induced. If cavities were visualized, the pneumothorax was maintained; and if the lung appeared normal, it was allowed to reexpand.

The thoracoplasty was then done. The group in which pneumothorax was continued during the stages of thoracoplasty in the opposite lung taught us that the bilateral advanced case could tolerate surgery. It was but another step to do a

bilateral thoracoplasty where pneumothorax could not be induced on the contralateral side. The management of the bilateral case by surgery opens up a new field in thoracic surgery.

I wish to say this about the generosity of Dr. Coryllos in giving so much credit to the medical service for his success in thoracic surgery. In my many years of experience in the thoracic surgery clinics, I have found that wherever the surgeon decides to go unaided, there has been a great deal of fatality, and where the surgeon and the medical man have worked in unison, we have accomplished our best results.

DR. L. S. PETERS.—There are so few tuberculosis specialists in this audience that I think I have to come to the defense of them every now and then. First of all, let me state that I am a believer in bilateral compression therapy, but I think all men doing tuberculosis work, so-called internists, or whatnot, should hesitate to press bilateral compression to the extreme that it is being pressed in institutional work. I believe that all bilateral compression therapy should be done in institutions where the patients are under close observation by the internist and by the surgeon, and I think that tuberculosis specialists not connected with institutions should limit their work to the safer risks; and, when they have bilateral cases that need close supervision, should refer them to institutions where they can have that supervision.

I think that the combination of internist and surgeon, regardless of Dr. Lilienthal, is necessary to the best results.

DR. JOHN ALEXANDER.—In connection with Dr. Coryllos' and Dr. Ornstein's interesting presentation, it may interest you that Dr. John C. Jones and I recently reviewed the results in 100 consecutive cases of bilateral active tuberculosis in which bilateral collapse therapy, exclusive of unaided bilateral pneumothorax, had been used. Many combinations of operations were employed. Since the function of both lungs was extensively destroyed, thoracoplasty was avoided whenever possible because it permanently excludes a considerable portion of the lung from function.

Ninety-five of the 100 patients had bilateral cavities, and the remaining 5 had only unilateral cavitation. The results from six months to four years after operation are: arrested or apparently arrested (closed cavities and consistently negative sputum), 46 per cent; quiescent (negative sputum in almost all of this group), 26 per cent; improved, 11 per cent; unimproved or worse, 10 per cent; dead, 7 per cent.

ARTIFICIAL RESPIRATION BY AN APPARATUS WHICH PERMITS MEASURED AND CONTROLLED VOLUMES AND PRESSURES*

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EFFORTS to resuscitate victims of asphyxia who are apparently dead have been attempted in all ages. Some of the methods resorted to were most fantastic. There was, for instance, that method known as "resuscitation by fumigation," which consisted of distending the lower bowel with tobacco smoke. "Barrel rolling" was practiced for many years in cases of drowning.

The records of the last one hundred and fifty years reveal that ingenious devices and methods were invented and used with reported success, but were then found wanting and discarded. Foot bellows, single and duplex piston pumps for positive pressure and suction were used in the late eighteenth century. Insufflation of the lungs with air through face masks, tracheotomy tubes, intralaryngeal and even intratracheal tubes was practiced during that period.

During the past seventy-five years, the most valuable contributions, the postural methods and the principles of artificial respiration by the positive and the negative pressure methods, were developed.

In the apparatus demonstrated and described below, I have endeavored to embody the valuable principles evolved by the eminent physiologists and clinicians of the past, and I present it in the hope of making a further contribution to the technic of resuscitation by artificial respiration.

The cases that present for resuscitation may be classified into four groups, depending upon the degree of asphyxia. The degree of asphyxia is proportionate to the extent of the trauma inflicted by the asphyxial agent upon the brain and the vital respiratory and cardiac centers.

In the first group, the nervous system has been but slightly traumatized. The patient is suffering from more or less shock. He may or may not be conscious, but the voluntary or reflex respirations have not been abolished. These patients require no artificial respiration and are usually revived by mild resuscitative measures, such as the application of heat, stimulants, inhalation of oxygen, or of a low mixture of carbon dioxide (7 per cent) and oxygen (93 per cent).

*Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery at New York City, June 3, 4, 5, 1935.

At the other extreme is that group in which the brain and the vital centers have been so intensely traumatized by prolonged and profound asphyxia that the patients are beyond any known human efforts to save them. These patients inevitably die.

Between these two extremes there is one group of asphyxiated patients in whom there is a temporay suspension of animation. There is more or less paralysis of the respiratory and cardiac centers. There are no visible respiratory movements. The heart sounds are inaudible, and the pulse is imperceptible. The vitality of the nervous system, however, is not completely abolished, and is still capable of recuperation. Nature has liberally provided normal life with "factors of safety," which account for the revival and survival of these patients, no matter what resuscitative technic is employed. Any method of artificial respiration or any apparatus for that purpose is sufficient.

Then there is that tragic group of profoundly asphyxiated patients who are the physician's greatest concern. The respiratory and cardiac centers are almost completely paralyzed, and life is at its lowest ebb. Though hope is not completely gone, these cases unfortunately are usually fatal. They may possibly be saved by more effective resuscitative measures, and particularly, more efficient measures of artificial respiration, supplemented, when necessary, by cardiac massage, intracardial medication or electrical stimulation. Success or failure in the resuscitation of these extreme cases depends, in great measure, upon the technic employed.

Effective artificial respiration depends on successful ventilation of the lungs. The problem then arises: What volumes and what pressures are essential?

Under normal conditions there is a respiratory exchange in the average adult of approximately 500 c.c. of air during each quiet respiration. By voluntary forced inspiration, the tidal volume may be increased by approximately 1,500 c.c. of complemental air. A supplemental volume of 1,500 c.c. of air can be exhaled by voluntary forced expiration. The vital capacity of the average adult lung, therefore, totals approximately 3,500 c.c.

I contend that in extreme cases of asphyxia, with respirations at a standstill and the vital centers in the medulla almost completely paralyzed, insufflation of small volumes under low pressures may not suffice to inaugurate that chain of stimuli which motivate the respiratory mechanism. Bolder methods must be employed. Two or even three times the normal tidal volume may be necessary and may be insufflated without danger.

The intrapulmonary pressure, which, under normal conditions, ranges between approximately minus 3 mm. of mercury at the end of a quiet inspiration and approximately plus 3 at the end of a quiet expiration, may be raised with these extra volumes to as much as plus

20 mm. or more of mercury. These figures, offhand, may appear excessive, but not when we realize that during violent coughing, singing, sneezing, or blowing, the intrapulmonary pressures rise to more than twice this tension.

Any apparatus used for artificial respiration should, therefore, be equipped to deliver at each inspiration ample measured and controllable volumes which will effectively inflate the lungs to the desired capacity within safe and adjustable pressure limits. To this end and purpose the apparatus described below was developed.

In extreme cases, simple inhalation methods, without supplemental measures to carry the resuscitative gases to the vital centers, are obviously useless, and nothing more need be said on this point.

The postural methods, of which the Schaefer prone pressure technic is the most popular, are used extensively with reported success. By actual measurements, however, these cannot effect a respiratory exchange of more than 250 to 350 c.c. per respiration. These amounts are less than the normal tidal volumes of the average adult and may be ineffectual.

Various inhalators commonly used to supplement the postural methods and which rely upon a face mask and resilient bag containing resuscitative gases are also ineffectual in extreme cases. Sufficient pressure to inflate a lung with the necessary volumes cannot be developed in a resilient bag, even at its maximum inflation. Encasing the bag in a cord network yields slightly better results; but, in either event, the volumes and pressures remain unknown quantities.

The lung motor and the pulmotor offer greater volumes and higher pressures. The lung motor provides for active expiration by suction, and the pulmotor, in addition, provides for automatic changes between inspiration and expiration. The principles upon which they were built and the features they incorporate have been condemned by a commission on resuscitation appointed by the government, for two reasons: first, because the mechanism by which the automatic change from inspiration to expiration and vice versa is accomplished depends upon a definite prearranged and invariable pressure limit at the delivery end, i.e., the patient. Any resistance in the respiratory tract to the flow of the resuscitating gases will raise the insufflation pressure to its predetermined limit. This automatically changes the inspiratory to the expiratory phase *prematurely,* and little or no resuscitating gases will reach the alveoli.

The second objection is the suction element for active expiration. Suction closes and obliterates the thin-walled bronchioles before it succeeds in emptying the alveoli of their noxious or poisonous gases. This feature thus defeats the very purpose for which artificial respiration is intended. The same criticism applies to any apparatus used for artificial respiration which is built on these principles.

The Drinker apparatus, the outgrowth of the Sauerbruch negative pressure chamber, maintains artificial respiration essentially on the principles of negative pressure. Since the rhythm, rate and amplitude of the induced respirations can be controlled by the operator, this machine is very valuable for effective artificial respiration. For prolonged artificial respiration, it is probably the most efficient method available. It has, however, several drawbacks. Its size makes it not practicably portable. It requires several persons to transfer the patient to the apparatus. The very nature of the negative pressure principle by which it functions rules it out as a resuscitating machine in the operating room during laparotomies, unless the abdomen is quickly closed to avoid evisceration.

The apparatus described below is a light portable machine, which I designed for artificial respiration with measured and controlled volumes and pressures and for inhalation therapy. It is designed to deliver either the tidal volume or double and triple the tidal volume with each inspiration; to indicate visibly the intrapulmonary pressure established at all times; and to guard against excessive intrapulmonary pressure. Oxygen, low and high percentages of carbon dioxide oxygen, or other resuscitative and therapeutic gases, or even air may be employed. It is adaptable for administration by a face mask, intrapharyngeal airway, or intratracheal tube.

The apparatus incorporates the following features:

A manifold (A) with three yokes or inlets to accommodate one cylinder of oxygen, one cylinder of a low percentage mixture of carbon dioxide and oxygen, and one cylinder containing a higher percentage mixture of carbon dioxide and oxygen or other therapeutic gases. Each gas inlet is supplied with an individual shut-off valve which permits the installation of fresh supply cylinders without halting or interfering with the resuscitative measures. The apparatus can be adapted to, and used with, any size medical or commercial cylinder.

A pressure gauge (B), located on the manifold, indicates the available supply of gas in any one cylinder.

An air inlet (C), guarded by a *stopcock,* permits the entrance and use of air from a foot bellows, air compressor, or from a central supply, in the absence of other resuscitative gases.

A pressure regulator and reducing valve (D), especially constructed, delivers from 35 c.c. to 600 c.c. of gas per second (at ordinary temperature and barometric pressures), which comprise the range of the average tidal volumes for the various age groups, listed below:

Birth	35 c.c. per inspiration
1 year	65 c.c. per inspiration
3 years	80 c.c. per inspiration
5 years	110 c.c. per inspiration
10 years	250 c.c. per inspiration
15 years	350 c.c. per inspiration
Average adult	500 c.c. per inspiration
Large adult	600 c.c. per inspiration

A flow gauge and *dial* (E), surmounting the regulator, indicate the volume flow per second and the average tidal volumes.

A control valve (F) permits the operator to regulate and set the amount to be delivered for each inspiration.

Thus, either the tidal volume or double the tidal volume for any age group can be delivered with each inspiration lasting two seconds. Triple

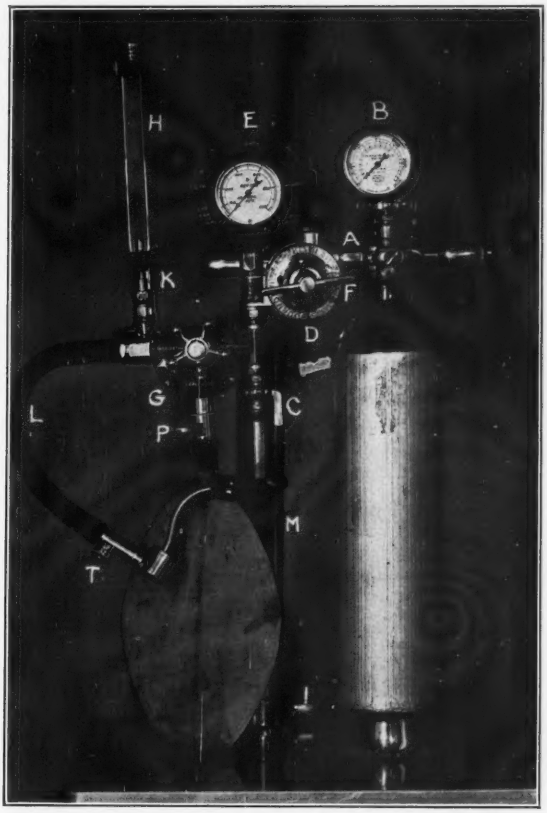

Fig. 1.—Apparatus for artificial respiration with measured and controlled volumes and intrapulmonary pressures.

the tidal volume, if necessary in extreme cases, can be delivered with each inspiration by maintaining the inspiratory phase for three seconds.

A four-position respiratory valve (G), controlled by the operator, regulates the rate and rhythm of the respirations and the amplitude of each inspiration and expiration. An arrowed pointer indicates the various positions of the valve: The inspiratory position permits the resuscitating gases to flow from the apparatus direct to the patient without an intervening bag. The expiratory position shuts off the

supply to the patient and permits the expired gases to escape into the open air. The inspiratory phase is an active process on the part of the machine and the operator, who aims to inflate the lungs to the desired capacity and pressure; the expiratory phase is passive. Any suction to facilitate expiration is unnecessary, even dangerous, and is intentionally dispensed with. The natural high elasticity and resiliency of the lungs affect the immediate recoil to the collapsed state when the pressure or inflating force is removed at the end of the inspiratory phase. The inhalation position is used only when there are reflex or natural respirations, and permits to-and-fro respirations of any therapeutic gases from a bag. The extent of rebreathing is regulated by an expiratory valve (T). A butterfly air vent (P) at the entrance to the bag permits respirations of air. The neutral position shuts off the supply from the apparatus and prevents escape of the insufflated gases from the lungs. The mercury in the manometer (mentioned below) will cease to oscillate and will register the intrapulmonary pressure.

A manometer (H), located on the patient's side of the respiratory valve, indicates the intrapulmonary pressure established at any phase of the respiratory cycle. The intrapulmonary pressure can thus be observed and read at all times.

A large dial safety valve (K) also located on the patient's side of the respiratory valve automatically blows off when the intrapulmonary pressure exceeds the safety limits. The relief valve can be adjusted to blow off at various pressures, from 5 mm. to 20 mm. or more of mercury. As a further precaution against the danger of excessive intrapulmonary pressure a second safety valve is incorporated. This is of the mercury type and can also be adjusted and set to blow off at various pressures.

A delivery tube (L) connects the apparatus with the patient. It is short (to eliminate dead space), flexible, noncollapsible, and of large lumen, which permits to-and-fro breathing without resistance. The tube ends in an adaptor which fits a face mask, a pharyngeal airway, or an intratracheal tube.

A manually operated suction pump and *receptacle* (M) maintain the oral and respiratory passages clear of obstructing fluid or semifluid material.

The main part of the apparatus is supported upon a telescoping upright mounted on a movable base.

As mentioned above, the apparatus is adapted for the administration of artificial respiration by a face mask, a pharyngeal airway, or an intratracheal tube.

Unquestionably, artificial respiration by intratracheal insufflation is most effective, when conditions permit. Unfortunately, however, when the emergency arises, the services of a physician skilled in in-

tubation are rarely available, making this method unpractical. When this method is employed, however, it is very important to use a catheter which fits snugly within the trachea. Otherwise, the gases escape alongside, and all measurements and controls fall short of their intended purpose. An inflatable balloon cuff, at the end of a catheter within the trachea, or a firmly fitting gauze pack surrounding the catheter in the mouth will minimize leakage.

A well-fitting face mask is less efficient but more practical because it can be applied by a lay rescuer, who is usually first on the scene. Distention of the stomach must be avoided either by pressure upon the epigastrium or by the insertion of a stomach tube. An airway, too, is indispensable to prevent intraoral obstruction by the falling back of the tongue.

A very practical, easily executed, and efficient method is the use of an airway with a special adaptor to fit the large calibred rubber tube leading from the apparatus. Gauze firmly packed around the tube within the mouth and a nasal clamp minimize leakage. The mask is eliminated altogether. The insertion of a large stomach tube prevents overdistention of the stomach, empties it of any toxic contents, and may be used to administer stimulating and nourishing liquids.

It has been suggested that artificial respiration may successfully be carried out by continuous intratracheal insufflations of air or oxygen. This procedure maintains continuous respiration without respiratory movement in a patient who is in otherwise good physical condition and in whom the vital centers are functioning normally. With a respiratory mechanism completely paralyzed, however, complicated by a failing circulatory system and life at a low ebb, merely keeping the lungs inflated may not suffice to restore respirations. To carry out successful artificial respiration, the object is to avoid keeping the lungs continually distended. By inflating and deflating the lungs, simulating the mechanics of physiologic respirations, one may succeed in inaugurating that chain of stimuli which may again establish voluntary breathing conditions. Rhythmic inflation and deflation of the lungs create variations in intrathoracic pressure, which have a marked influence upon the heart and intrathoracic vessels, and in turn a beneficial effect on the general circulation.

I am presenting this apparatus in the hope that a more effective technic for artificial respiration may be instrumental in saving the lives of those threatened by asphyxiation.

285 CENTRAL PARK WEST

DISCUSSION

DR. POL. N. CORYLLOS.—If I have correctly understood Dr. Branower, the apparatus which he has presented here performs insufflation of oxygen or O_2-CO_2 mixture under pressures and volumes which can be adjusted according to the needs. Its action is intermittent in order to allow expiration, and the number of interruptions

can be equally well regulated. Therefore this apparatus is similar to the pulmotor type of resuscitating machines which are not without danger for the lung unless they are used by very competent men. The regulation of the pressure under which gases can be insufflated into the lung is not an easy matter; it is for that reason that safety valves have been provided on Flagg's and other similar apparatus. I perfectly agree with Dr. Branower that there are cases of asphyxia in which respiration is completely arrested and in which manual artificial respiration does not suffice to insure the inhalation of an adequate volume of tidal air because in these cases the chest is often immobilized in marked expiratory collapse, caused by the suppression of muscular tonus. In these cases, as we all know, the most efficient method of resuscitation is the mouth-to-mouth insufflation, which allows the insufflation of the right amount of air, containing the right amount of CO_2 under a correct pressure and at the correct intervals of time.

On the other hand, manual artificial respiration is not always feasible in operative cases, and it is most often impossible in cases of thoracic surgery, in which we are more particularly interested here.

In all insufflation apparatus pulmonary ventilation is insured in an antiphysiologic way; air is not sucked into the lung because of the passive distention of the organ; the lung is forced to distend because the air that is pushed into it is under positive pressure, while for expiration we trust to the resiliency of the elastic elements of the lung and of the chest.

However undesirable may be this reversal of physiologic functions, it is the only procedure at our disposal in cases of complete apnea and marked respiratory collapse of the chest. None the less, these shortcomings may, I believe, be corrected to a great extent and the dangers of injury to the lung completely avoided by the use of an apparatus which insufflates gases under a steady pressure and which assists expiration by slight suction. Thus the volume of the gas insufflated each time becomes a function of the pressure; with pressure remaining at below 15 mm. of Hg, any injury to the lung becomes impossible. If now, as soon as this pressure is reached, the instrument automatically stops insufflation and produces a mild suction, 4-5 mm. Hg, then expiration is facilitated, the dead pulmonary space is decreased, the ventilation of the lung is improved, and the rhythmic changes of intrathoracic pressures, so helpful to the circulation, approach the normal.

I had the opportunity lately to experiment, in the laboratory and the hospital, with an apparatus which in my opinion fulfills the above mentioned requirements. This apparatus is known as the E and J Respirator. I shall quote only one experiment, which has impressed me; I was able with this apparatus to maintain alive, for over twelve hours, dogs with their chests widely open, with very little supervision. I have used this apparatus on two lobectomies in the human being and in a considerable number of experimental animals. However, I am not yet ready to express a definite opinion about it.

These remarks are not directed against the apparatus of Dr. Branower, which certainly has its merits. They are only made in order to stimulate the commendable efforts of this society for improvements of our methods for artificial respiration, differential pressures, and resuscitation.

DR. WILLIAM BRANOWER.—I experimented with an apparatus of the type mentioned by Dr. Coryllos on the excised lung and saw it used in the clinic in a lobectomy case during operation by Dr. Coryllos. The apparatus repeated in the operating room what I had observed in the laboratory. The inspiratory and expiratory phases succeeded each other automatically in very rapid succession, but neither the excised lung nor the patient's lung was inflated to any appreciable extent. It is hard to conceive how sufficient gas to inflate the lungs effectively could be delivered in the fraction of a moment consumed by the inspiratory phase.

Although that apparatus may serve a useful purpose in many instances, in my opinion it incorporates two features to which the commission on resuscitation took objection, namely, automaticity, and suction as an aid to expiration. The apparatus provides variable volumes, but the amounts delivered depend upon a prearranged, definite, and invariable pressure limit. When, from whatever cause, this pressure limit is reached in any part of the respiratory passages, the inspiration automatically changes to an expiration before the resuscitative gas has reached the alveoli. This accounts for the very rapid succession of the artificial inspirations and expirations.

The element of time must be considered in relation to the rate, rhythm, and amplitude of the respirations. When the governing respiratory center is not functioning, its regulation must be assumed by the person performing the artificial respiration. By means of the respiratory valve incorporated in the apparatus presented today, he can regulate the duration of each inspiration and each expiration, as well as the rate and rhythm of the respirations.

Suction as an auxiliary to expiration is unnecessary, but may be used only to maintain patency of the air passages. Suction contracts the trachea and bronchi and may imprison the noxious gases within the alveoli.

Unless a snug fitting intratracheal catheter can be quickly and immediately introduced, it is advisable to depend upon a pharyngeal airway and a well-fitting face mask to deliver the resuscitating gases to the lungs.

THE VAGUS AND ITS RELATION TO THE SURGERY OF THE LUNG

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INTRODUCTION

THE literature contains many records of a fatal collapse during a major intervention on the lung, of which the following is typical. Denk describes an operation for bronchiectasis in which he was carrying out the cross resection of Brauer. The general condition of the patient was good but suddenly, just as a suture had been passed through one of the larger bronchi, he collapsed and died. At autopsy no mechanical cause for death could be demonstrated, and Denk, concluding that this was a "reflex" death, recommends that as an invariable preliminary to lobectomy a block of the vagus nerve should be carried out. (A convenient technic for the injection of the vagus on its emergence from the jugular foramen has been elaborated by Heidt.)

It is not always possible to accept the reflex explanation of this and similar incidents. Gross air embolism such as may prove fatal after a wound of one of the large veins at the root of the neck is easy to recognize at autopsy, as death results only after aspiration of a large quantity of air which is found in the right heart and in the pulmonary circuit. On the other hand, a laceration of the lung involving a branch of the pulmonary vein may be followed by the aspiration of air into the left auricle, and the distribution of even a minute quantity into the systemic circulation may cause death by occlusion of a coronary artery or one of the bulbar vessels. The recognition of a minute air embolism at autopsy offers great difficulties—so great that Hoffheinz, who has described in great detail the precautions necessary in carrying out a postmortem examination in suspected cases, concludes that under certain circumstances it is impossible.

The patient, by the time he has reached the thoracic surgeon, has frequently a myocardium which has been exposed to toxic influences over a long period, and sudden death may simply be the failure of an inefficient heart to withstand the operative trauma. An exact analysis of the events immediately preceding death is seldom possible under the conditions governing operative surgery. No convenient mechanism exists for obtaining a continuous record of respiration and circulation of the operated subject, so that it is often impossible to decide even the elementary question as to which of these systems failed first, and unless by

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chance the heart happens to be exposed in the operation field a decision as to whether the fatal event was fibrillation or simple cessation of cardiac action is impossible. It is therefore necessary to turn to the experimental animal, which lends itself to dispassionate and accurate observation.

There are certain inherent difficulties in the use of the experimental animal. Thus of a score of workers only Lewis and Capps (1904) have produced sudden death with any degree of consistency, although more recently Sunder-Plassmann has reported occasional fatal collapse following his method of stimulating the bronchus from its lumen. In my own series of experiments an actual inhibition of cardiac action as a result of a mechanical stimulus was never obtained, and indeed it seems probable that the statement by Levy in 1913: "Permanent inhibition of the mammalian heart by reflex vagal section is unknown to mammalian physiology," remains true today. At the same time it was possible to demonstrate cardiac and respiratory effects of reflex origin. It is perhaps inevitable that in any investigation involving the autonomic system there should be some inconsistency. The reaction of the somatic nervous system is standardized—stimulation of the sciatic nerve will always give a contraction of the flexor muscles of the thigh—but many conditions govern the response of the autonomic nervous system, and a full understanding of these conditions is not yet in our possession. Thus stimulation of the vagus in the neck may slow the heart, increase its rate, or leave it unaffected. This inconsistency is seen also in the clinical literature. Nissen (1931), who carried out the first successful pulmonectomy by allowing the lung to slough out after ligation of the pedicle, records that as a ligature was applied to the lung root there was temporary cessation of the heartbeat and for a time a fatal outcome seemed likely. On the other hand, Graham, who performed the first successful pulmonectomy in one stage some two years later, records that pulse rate and respiration remained unaffected. Rienhoff (1933), who did two successful one-stage pulmonectomies, also records that the pulse and respiration rate were unaffected. In 1926 Morrison reported an interesting series of total pulmonectomies in the rabbit. He found that the rabbit survived total excision of the lung only if the vagus stem were divided in the neck as a preliminary to the intervention. Working on the baboon, the cat, and the rabbit in the Sudan, I found that these animals were in no way disturbed by the operation of excision of the lung with a preliminary vagotomy, but at this time I did not carry out a control series of experiments. Morrison and I worked with an open pneumothorax and in this respect differed from all other experimentalists in this field, who since 1904 have used some form of differential pressure anesthesia. This point was made by Morrison in his paper. More recently I have repeated a series of pulmonectomies in the cat, the pig, and the rabbit, with an intact vagus but employing positive pressure anesthesia, and have experienced no immediate mortality.

THE EXPERIMENTS

In the experiments recorded here the lung root was selected as the site for investigation. In the cat, the dog, and in man the lung root possesses a very definite posterior plexus to which the vagus contributes strong branches, easily recognizable to the naked eye, as the main stem descends to the esophagus. An anterior pulmonary plexus is usually described but in practice does not exist. There are fine nerve fibers, running mainly with the pulmonary veins, which have their origin in the deep cardiac plexus and have no connections with the vagus stem.

Dogs were the subject of experiment and were anesthetized by morphia and nembutal (a 10 kilo dog had 2 gr. morphia and 4.5 gr. nembutal). In a few experiments cats, anesthetized with chloralose (0.6 gr. per kilo) were used. After the chest was opened, oxygen was given by the Tiegel-Henle positive pressure apparatus, and respiration and blood

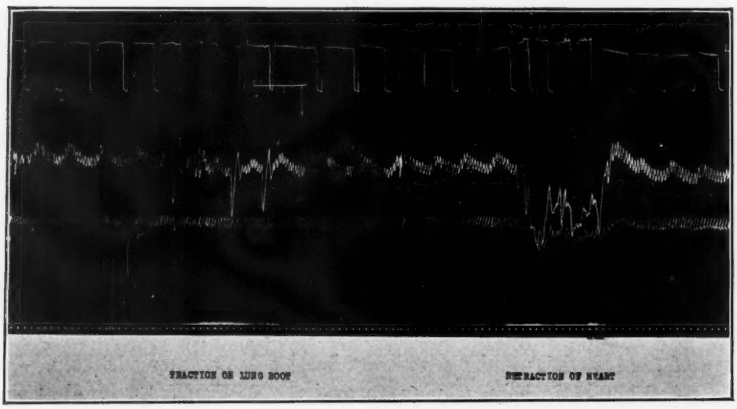


Fig. 1.—Showing effect of traction on the lung root in dog. Morphia gr. 2 and nembutal gr. 4. Upper curve is record of respiration; middle curve is record of blood pressure; lower curve is record of pulse. Time, ten seconds.

pressure were recorded in the usual way. An ingenious modification of the McDowall cannula by Mr. F. Watson of the Buckston Browne Surgical Research Farm permitted the inclusion of a membrane manometer in the blood pressure circuit, and in this way the pulse record was obtained.

The effect of traction on the lung root of the dog under the experimental conditions is shown in Fig. 1. There is an alteration in the respiratory and cardiovascular records. In order to separate these two effects in subsequent experiments, electrical stimulation of the anterior surface and of the posterior surface of the lung root was substituted for mechanical stimulation. For this purpose an ordinary induction coil at 11 cm. distance was used.

Stimulation of the posterior aspect of the lung root caused alteration in breathing. At times a period of actual apnea was produced and in

other experiments a difference in the rate and character of the respiration could be recognized (Fig. 2). Stimulation of the anterior surface of the lung root caused an increase in pulse rate, an irregularity in cardiac action, and a fall in blood pressure (Fig. 3). These effects could be obtained through an intact pleura but were accentuated if the pleura was incised and the electrode applied to the subpleural tissues.

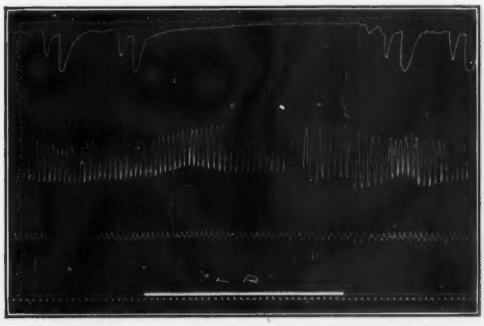


Fig. 2.—Showing respiratory reflex in dog. Morphia gr. 2 and nembutal gr. 4. Signal, stimulation of posterior aspect of lung root. Upper curve is record of respiration; middle curve is record of blood pressure; lower curve is record of pulse. Time, ten seconds.

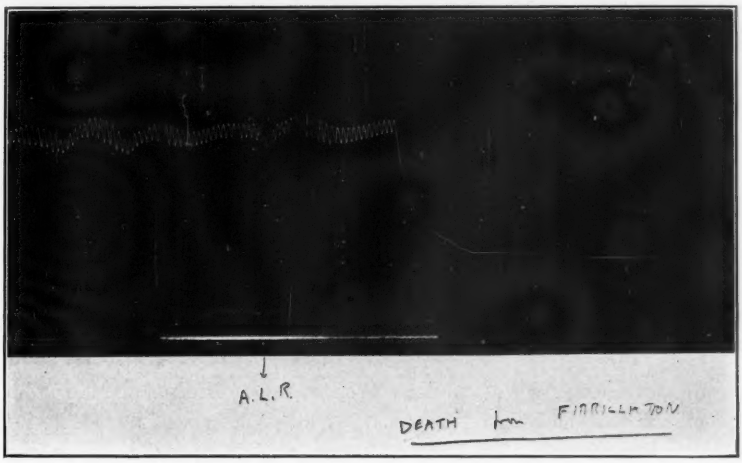
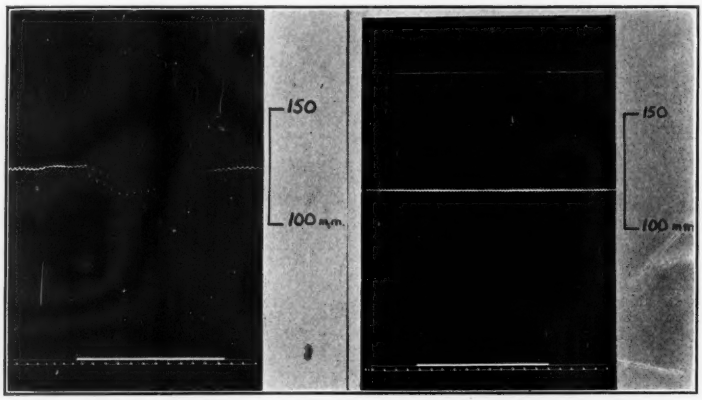


Fig. 3.—Showing fatal cardiovascular reflex in dog. Morphia gr. 2 and nembutal gr. 4. Signal, stimulation of anterior surface of lung root. Note that circulatory failure occurs before respiratory failure.

A method having been devised by which constant reflex effects could be obtained, various procedures were adopted in order to prevent their appearance.

Section of the vagus in the neck or just above the lung root abolishes the respiratory reflex but leaves the cardiac reflex unaffected. Infiltra-

tion of the stellate ganglion and upper dorsal sympathetic chain leaves both reflexes unaffected. The application of a swab soaked in 1 c.c. of 15 per cent cocaine for a period of five minutes will render both areas insensitive but in the dog at the cost of a fatal decline in the blood pressure, presumably the result of absorption. Ten per cent cocaine applied in the same way and for a similar period will render the anterior lung root anesthetic but not the posterior lung root; deleterious effects from its absorption were not observed. Percaine in 5 per cent solution failed to render the area insensitive, and deleterious absorption effects were observed. An injection of 1 per cent novocaine beneath the pleura covering the lung root abolished both respiratory and cardiovascular effects. It has been established that atropine in very large doses—up to 11 mg. per day—can be tolerated by man, and it was long ago suggested (Grave, 1914) that a large dose of atropine might protect a



A. *B.*

Fig. 4.—Showing effect of spinal anesthesia in cat. Chloralose. Signal, stimulation of anterior surface of lung root. *A*, Before spinal anesthesia. *B*, After spinal anesthesia. Novocaine 50 mg. intrathecal. Time, ten seconds.

patient during chest operations. In my experiments it was found that atropine, even pushed to a dangerous limit (5 mg. for a dog), failed to diminish the intensity of the lung root reflexes. In the cat total spinal anesthesia may be induced by the injection of 30 mg. of novocaine into the lumbar subarachnoid space. In such an animal there is a marked fall in blood pressure and respiration ceases. The supply of oxygen through the Tiegel-Henle apparatus maintains life, and after a period of about thirty minutes the animal resumes normal respiration. It is clearly impossible to observe the effects of stimulation of the lung root on the respiration in such an experiment, but it can be shown that cardiovascular effects are no longer produced by stimulation of the anterior surface of the lung root. In some earlier experiments it seemed possible that this immunity was due to the lowered blood pressure but,

as is shown in Fig. 4, the effect persisted even when the blood pressure had been restored to its original level by the injection of adrenalin.

It is generally believed that an intervention on the right chest is fraught with greater danger than similar intereventions on the left chest. Kusakabe has recently claimed that the nervous connections between the right lung root and the cardiac plexus are more intimate than those on the left side, and in his experiments stimuli of equal intensity produced more marked effects when applied to the root of the right lung. My own findings failed to confirm this view—no special sensitivity of the right root area was demonstrated.

CONCLUSION

In the course of this work some observations were made on the sensitivity of the lung root in man. During an intervention on the thoracic esophagus, a patient whose general condition had remained good despite a wide exposure of the mediastinum manifested great distress as a separation of the esophagus from the left main bronchus was attempted. As soon as the attempt was abandoned the general condition returned to its former state, and recovery from the exploration was uneventful. In three cases the lung root was explored and a ligature of the pulmonary vein after the manner of Kerschner carried out. No cardiac or respiratory distress was observed except in one case, in which retraction of the heart by the hand of the assistant produced a marked but temporary bradycardia. In these cases a swab soaked in 15 per cent cocaine was applied to the area without harmful effects, but, in view of my experimental findings, in future some other method of local anesthesia will be adopted. During an operation for the open division of adhesions between the lung and the thoracic wall, the patient being anesthetized by evipan, I had occasion to observe that the region of the lung root was highly sensitive.

The conclusions reached in the main confirm those of Heller and Weiss. The vagus is an important sensory nerve, and it is an advantage to prevent nociceptive impulses from passing along it. At the same time, blocking of the vagus stem is not a safeguard against reflex disturbances while operating in especially sensitive regions such as the lung root; respiratory reflexes are certainly abolished, but cardiovascular reflexes remain. Safety can be attained only by a local infiltration of the operation field. A swab soaked in 1 c.c. of 10 per cent cocaine is innocuous and will at least protect against cardiovascular effects; cocaine in higher concentration is probably dangerous. Large doses of atropine are without value. My experience with total spinal anesthesia in man is recorded elsewhere. It proved a satisfactory anesthetic for some operations on the chest wall such as thoracoplasty, but it was not used in any operation on the lung. The experimental results recorded above suggest the trial of total spinal anesthesia for intrathoracic operations.

The experiments recorded in this paper were begun while the author was the College Research Scholar, and all of them were carried out at the Buckston Browne Research Farm; the author wishes to express his gratitude both to the Council of the Royal College of Surgeons of England and to the Medical Research Council, from whom he is now in receipt of a personal grant. A preliminary communication was made to the British Association of Surgeons in May, 1934.

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ACUTE EMPYEMA THORACIS*

A Study of Healing and Pulmonary Reexpansion

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INTRODUCTION

THE treatment of pleural suppuration by providing drainage with the thrust of a knife or cautery was known to Hippocrates. Yet, more than two thousand years later, there was so little confidence in the surgical management of empyema that when Dupuytren,[35] the ablest French surgeon of his time, was stricken with this disease (in 1835) he refused operation with the remark that he would "rather die by the hand of God than with the help of a physician."

One hundred years after Dupuytren the treatment of empyema can be approached with hopefulness rather than despair. The mortality has been reduced, the period of healing shortened, and chronicity, to a large extent, prevented. These results have been achieved despite the lack of complete accord concerning either the theoretical principles involved or the practical application of methods of treatment.

It is fundamental to regard empyema not merely as an example of suppurative inflammation, but as an accumulation of exudate in a body cavity in which contact of the opposed serous surfaces or the maintenance of a negative pressure is necessary to the normal performance of a vital physiologic function. Evacuation of pus, therefore, must be conducted in such a manner as to avoid further embarrassment of respiration, and to provide for early restoration of the natural anatomic relation of the elastic lung to the rigid thoracic wall.

Principles of treatment have been outlined somewhat differently by Immerman,[68] Graham,[48] Ochsner,[95] and Harrington,[54] but those listed by Harrington are sufficiently inclusive to cover the salient points referred to by the others. They are as follows: (1) adequate drainage; (2) avoidance of open pneumothorax; (3) rapid sterilization of the diseased area; (4) early obliteration of the cavity by expansion of the lung; and (5) good medical care with particular attention to the nutrition of the patient. The fifth of these principles may be dismissed without discussion. Reference will be made here especially to those problems which are peculiar to the surgical treatment of empyema. The first four principles of Harrington are, therefore, rearranged and restated to serve the

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purposes of this review, special attention being devoted to the problem of reexpansion of the lung and healing of the empyema cavity.

AVOIDANCE OF OPEN PNEUMOTHORAX

The studies of Graham and Bell on open pneumothorax and Graham's further writings on this subject[46-50] are so well known as to require little comment. It was shown that in a normal animal a change of pressure occurring in one pleural cavity is reflected in the other, and that the size of the opening in the chest which an individual can withstand is governed to a large extent by his vital capacity. Other changes, such as heat loss and circulatory disturbances, may occur as a consequence of open pneumothorax, but when death results it is usually due to asphyxia.

The practical application of the physiologic principles discovered by Graham and Bell is to be found in the surgical principle of avoiding open pneumothorax in the early stages of the treatment of empyema. Unfortunately the fundamental principle involved has often been submerged in debate concerning the nature of the mediastinum of experimental animals. This surgical principle does not rest upon the results of animal experimentation alone but is based upon the clinical observation of competent surgeons that following open thoracotomy a tragic train of symptoms may sometimes develop, namely, cyanosis, dyspnea, even death. Magie[54] as early as 1908 appreciated the danger of immediate radical operation in acute empyema involving the greater part of the pleural space. During the World War dyspnea, cyanosis, and death were frequently observed to be the immediate consequences of open thoracotomy performed for drainage of the thin pus of the post-influenza streptococcic empyema prevalent at that time. It is the experience of Heuer[64] that the same unhappy result may occasionally occur incident to open drainage of a pneumococcic empyema. The guiding principle, according to Graham, is to delay open drainage of the chest until the pus is thick, indicating stabilization of the mediastinum and the formation of visceroparietal pleural adhesions.

ADEQUATE DRAINAGE

It is sometimes assumed that, aside from the considerations of open pneumothorax, the ideal time for drainage must be the earliest possible moment after the pleural effusion has been detected. On the other hand, Soresi[123] and Lockwood[76] have pointed out that the pleural effusion is nature's attempt to control infection. They believe the effusion is beneficial rather than harmful; and, therefore, before the empyema is encapsulated, fluid should be removed only if required for the relief of respiratory embarrassment. Lockwood is opposed even to the use of aspiration in the early stages except to relieve dyspnea.

Statistical data referred to in a previous article (Carlson and Bowers[23]) indicate that the mortality of empyema is determined largely

by the virulence of the organism, the age of the patient, complications, and the nature of the primary disease. There do not appear to be any decisive experimental or clinical observations either in support of the opinion that early aspiration is harmful or in support of the contrary view that early drainage is imperative. The published results in two series of empyema in children treated by McEnery and Brenneman[82] (chiefly aspiration) and Graham and Berck[51] (aspiration followed by open thoracotomy) teach that, in the incipient stages, aspiration is usually a satisfactory procedure.

It is difficult to learn from the literature or from personal experience what constitutes adequate drainage. One or more aspirations may lead to healing; drainage by means of an intercostal catheter frequently suffices; a bronchopleural fistula may provide adequate drainage; and occasionally spontaneous healing may occur without any form of drainage. On the other hand, many cases of empyema, particularly those due to the pneumococcus characterized by the formation of pus rich in fibrin, fail to make satisfactory progress until a large opening for drainage is provided. Obviously a decision as to the adequacy of drainage must be made in each case on x-ray evidence, signs of sepsis, and the tendency of the cavity to heal.

STERILIZATION OF THE EMPYEMA CAVITY

Sterilization of the empyema cavity was the primary objective under the regimes recommended by Lister,[75] Murphy,[92] and Hathaway.[57] Murphy aspirated pus and injected a few ounces of 2 per cent liquor formaldehyde in glycerin. The effusion eventually became a sterile serosanguineous fluid which was permitted to remain until absorbed. Healing might require from three to thirty months, an excessive period of healing according to modern standards.

Attempts to sterilize the cavity by means of antiseptics are now considered secondary in importance to drainage; but saline, Dakin's solution, Hermansdorfer's solution, antiseptic dyes, bile salts, and other irrigating solutions are frequently employed to remove pus and fibrin, to maintain the patency of drainage tubes, and to aid in sterilizing the cavity. Rea and Wangensteen[104] have recently made in vitro comparisons of Dakin's solution, trypsin, pepsin, sodium ricinoleate, and Hermansdorfer's solution, and conclude that Dakin's solution best meets the needs of a satisfactory irrigating solution because of its action in digesting pus and fibrin as well as because of its germicidal properties. Harrington compared saline, mercurochrome, and Dakin's solution in clinical cases and found no marked advantage of one over the others, although he preferred to use the latter. Gay and Morrison[42] were unable to establish the value of various antiseptic dyes in streptococcic empyema in rabbits.

Some surgeons (Heller,[62] and Douglas,[34]) consider irrigating solutions to be of no value, and the published reports of their cases treated without irrigations demonstrate at least that such solutions are not indispensable in the treatment of empyema.

It is obvious that localization and sterilization of a pleural infection may occur as a result of a natural protective mechanism without the aid of antiseptics, even without the aid of drainage. The importance of clasmatocytes (Gay and his coworkers[40-43]) and of polymorphonuclear leucocytes (Steinberg and Snyder,[124]) have been stressed, but Opie,[96] Cannon,[18-21] Rich,[108-109] Pacheco,[97] and their associates are of the opinion that humoral antibodies rather than cells of inflammation are of primary importance in the protection of immune animals. Rich and McKee injected pneumococci intradermally into immunized leucopenic animals and found that the organisms were held fixed at the site of injection for hours after control animals had died of septicemia. If a few leucocytes appeared in the region of the infection, bacteria were rapidly ingested and the lesion was sterilized. These experiments indicate that in immune animals humoral antibodies may be effective before mobilization of phagocytes has occurred.

The extent to which antiseptic solutions may aid this protective mechanism in sterilizing the empyema cavity remains undetermined. Possibly their greatest usefulness is in washing out pus, fibrin, and necrotic tissue and in maintaining the patency of the drainage tubes.

REEXPANSION OF THE LUNG AND HEALING OF THE CAVITY

The process by which the collapsed lung expands to fill an empyema cavity and the related but not necessarily identical process by which the cavity heals, provoked much discussion during the years immediately following the introduction of the operation of rib resection. Although much of the literature published on this subject appeared before the turn of the century, there is no unanimity of opinion today regarding the relative importance of the several factors alleged to bring about reexpansion of the lung and obliteration or healing of the cavity.

In attempting to formulate a definite opinion on this subject it is helpful to recognize that the process of healing and of reexpansion of the lung may vary somewhat according to the method of treatment employed. A review of clinical observations and experimental studies relating to this subject should also be of value.

Roser[111, 112] as early as 1864 commented on the ability of the lung to reexpand and in 1878 advanced the opinion that after opening the chest reexpansion occurred as the result of the formation of adhesions between the visceral and the parietal pleurae beginning at the hilus and extending peripherally. According to this conception the attachment of the lung to the chest wall permitted some expansion on inspiration. Contraction of young connective tissue and further extension of the angle of

fusion continued progressively until there was eventually a complete obliteration of the entire pleural space. Of necessity the lung had reexpanded to the chest wall. He assumed further that the connective tissue uniting the two surfaces finally was absorbed, permitting the lung to move freely again within the pleural space. Andrew H. Smith[121, 122] as early as 1869 appears independently to have reached conclusions regarding healing and reexpansion of the lung essentially the same as those of Roser.

The idea of a progressive obliteration of the pleural space bringing about expansion of the lung was opposed by Schede,[115] who denied that either the visceral or the parietal pleura was capable of producing granulations. Billroth[6] among others, however, supported Roser, and today the formation of fibrous adhesions between the parietal and the visceral pleurae is so generally admitted as to require no discussion.

Weissgerber[132] opposed Roser on other grounds. He contended that reexpansion of the lung by progressive adhesion of the visceral and parietal pleurae was impossible because the lung in empyema was collapsed; and, therefore, if adhesions formed, they would unite parts of the visceral and parietal pleurae not normally opposed. He argued that the result would be the adherence of a collapsed lung to only a part of the chest wall so that eventually there would remain a large parietal pleural surface, a much smaller visceral surface, and a residual cavity. He proposed the physiologic explanation that the intrapulmonary pressure on inspiration was negative but on expiration positive, and that the positive pressure on expiration was greater than the negative pressure on inspiration. This relationship, he believed, led to a progressive expansion of the lung.

Bouveret,[10] Schede,[115] Gerhardt,[44] and others had observed that in the presence of an open thoracotomy, cough or forced expiration caused the lung to expand. In some instances the lung was made to prolapse through the thoracic opening. But Aufrecht,[4] although admitting the possibility of expanding the lung by forced expiration, observed the movements of the lung through an open thoracotomy and found that with normal respiration progressive expansion did not occur. During inspiration he observed the lung to "sink in," during expiration to attain a volume about equal to that of the previous expiration. He pointed out that progressive expansion of the lung as a result of normal respiration was, in fact, excluded because the intrapulmonary pressure developed on expiration (2 to 3 mm. Hg*) was not sufficient to overcome the elastic contractile force of the lung (6 mm. Hg*).

These observations had been made with a relatively large opening in the chest. On the other hand, according to Aufrecht, when the diameter of the thoracic wound was less than that of the main bronchus, expansion occurred with each inspiration. In order to maintain the expan-

*Quoted from Donders.[33]

sion gained in this manner, a second factor was necessary, namely, fibrin formation so that agglutination of the lung and chest wall could occur.

Martens[86] studying experimental empyema in rabbits found that with a true open pneumothorax, the lung not only failed to expand but that if the condition was allowed to persist the lung was eventually fixed by fibrosis of the pleura and permanently lost its ability to expand.

Reineboth[105] (1897) planned a series of experiments on normal rabbits to gain further information regarding the mechanism of lung expansion. He prepared pleural windows by carefully removing the intercostal muscles, leaving the pleura intact. He then observed the movements of the lung through the pleural window after making a small or large opening in the chest wall. His observations may be summarized as follows:

With the diameter of the opening in the chest definitely smaller than that of the bronchus—

1. No matter how small the opening in the chest is, the lung collapses. The smaller the opening, the slower is the collapse.
2. Volume changes of the lung follow the chest wall; expansion on inspiration, deflation on expiration.
3. The degree of movement of the lung is in inverse proportion to the size of the opening.
4. Movements of the lung continue after the lung is collapsed.
5. If one clamps the trachea at the end of inspiration, the collapsed lung is expanded as a consequence of the ensuing forced expiration.

With the diameter of the thoracic opening definitely greater than that of the main bronchus—

1. No movement of the lung is noted with normal respiration.
2. With forced inspiration there is collapse of the lung; with forced expiration, expansion of the lung.

In other words, he concluded that inflation of the lung occurred on forced expiration regardless of whether the thoracic opening was larger or smaller than the main bronchus. Furthermore, with a large opening in the chest, volume alterations of the lung did not occur as a consequence of normal respiration; but with a sufficiently small opening, inflation occurred on inspiration, deflation on expiration.

Reineboth recognized that the size of the thoracic opening was only approximately related to the size of the main bronchus in the effects produced on movements of the lung. Graham[49] more recently (1925) has further elaborated this point. He states, "The ratio of the volume of air entering the chest through the thoracic opening to the volume entering the lung through the natural air passages would be equal to the ratios of their areas if it were not for two facts: (1) the natural air passages to the lungs have a considerable length which causes appre-

ciable friction and a resistance to the entrance of air; and (2) the natural elasticity of the lungs offers further resistance. On account of these two factors an opening in the chest wall of a somewhat smaller size than the narrowest part of the natural air passages will admit the same amount of air." Graham refers to the glottis rather than the bronchus as the narrowest part of the natural air passage.

Another observation made by Reineboth was that the dressing, particularly a moist, pus-soaked dressing, greatly modified the effects of an open pneumothorax. This observation confirmed the views of Bouveret, Schede, and Gerhardt. On the basis of pressure measurements in empyema cavities of human patients, Reineboth concluded that an occlusive dressing converted a large thoracic opening into a small one so that expansion occurred on inspiration. It also acted as a valve permitting cough or forced expiration to bring about a progressive expansion of the lung.

Martens (1898) similarly concluded from experiments on rabbits that a moist dressing aided in reexpanding the lung.

This conception of the valve effect of dressings is widely accepted. Witness the discussion of healing and reexpansion offered by von Eberts[128] in 1911, in which he also restates the fundamental teachings of Roser: "... the granulating pleurae in the perpheral zone of the cavity are brought together and adhere, granulous retraction further coapts the separated pleurae until the whole cavity is in the majority of cases finally obliterated, not because of the inherent tendency of the lung to expand in the face of atmospheric pressure, but because of the inherent tendency of granulating tissue to contract and because of periodic respite from atmospheric pressure afforded by the pus-soaked dressing." According to this conception, when an open thoracotomy wound is covered by a pus-soaked dressing, one is concerned no longer with a true open drainage but with a form of valve drainage of uncertain efficiency.

The foregoing discussion has dealt with the problem of expansion of the lung against the force of atmospheric pressure in the presence of an apparent or true open drainage. The mechanism of expansion in a closed drainage system or under the intermittent aspiration method of treatment is obviously a somewhat different problem. Sauerbruch,[114] although confessing some uncertainty concerning the principles of reexpansion in open drainage, states unequivocally that a closed siphon drainage is a hydraulic system with an artificial sucking action like that of the chest wall or diaphragm. The amount of negative pressure developed depends upon the height of the column of fluid in the siphon system.

Model thoraces have been constructed independently by Revilliod,[107] Douglas,[34] and Wangensteen[130] for the purpose of studying the principles of expansion of the lung. The model thorax consists of a rubber

balloon (elastic artificial lung) placed within a glass cylinder or bottle (rigid thoracic cage). The interior of the balloon is exposed to atmospheric pressure by means of a stem corresponding to the trachea. There is also a vent (thoracotomy) in the bottle to which any desired pressure may be applied. When this vent is open, the balloon rests in a position of collapse, the pressures on the inside and outside of the balloon being the same. If a suction is applied to the vent, the lung expands by virtue of the differential pressure created. The degree of expansion is dependent upon the relationship between the negative pressure employed and the elasticity of the model lung.

If the model chest is filled with fluid which is then siphoned off, the collapsed balloon expands; and if drainage of the fluid is complete, the model lung expands to the walls of the bottle, completely filling it. Douglas has pointed out that this process is the same whether the drainage is dependent or high up on the artificial thorax.

Wangensteen has stressed the relationship between elasticity of the lung and the degree of intrathoracic pressure. If the balloon is thick walled and difficult to distend, simulating a lung restrained by the thickened pleura of empyema, a high negative intrathoracic pressure may be created in the absence of satisfactory expansion of the lung. He concludes, therefore, that under such circumstances adequate drainage cannot be obtained by negative pressure alone but the injection of fluid or air will improve drainage by diluting or replacing the exudate.

Although it must be admitted that the model thorax does not reproduce all of the conditions of the living thorax in that respiratory movements are absent, it serves to illustrate the fundamental principles concerned in expansion of the lung under the conditions of open or closed drainage.

As aspiration is intermittent closed drainage, no special explanation is required for reexpansion under this form of treatment. It is axiomatic that, since the chest wall is rigid, when fluid is removed from the pleural cavity the displaced volume must be filled by fluid or gas by displacement of the mediastinum, by elevation of the diaphragm, or by expansion of the lung.

When air replacement is combined with aspiration, according to Danna,[31] gradual absorption of the remaining air in a large pneumothorax explains the reexpansion of the lung and the healing of large cavities; although he admits the possibility of closure of smaller cavities by cicatricial contraction of granulation tissue.

Bloch and Parrish[8] assert that with the aspiration-air replacement method, the pleurae are kept separated until the inflammation subsides; thus adhesions do not form or are slight in extent. They assume further that drains inserted into the chest are the cause of crippling adhesions which frequently lead to contraction of the thorax, loss of function, and severe pressure and traction symptoms.

Contrary to the opinions of Danna, and Bloch and Parrish, however, it is generally believed that healing occurs as a consequence of obliteration of the cavity by adhesion of the visceral and parietal pleurae independent of the method of drainage. This commonly accepted surgical opinion is based chiefly upon clinical observation. A few references may be found to necropsies performed upon patients recently cured of empyema (Gläser,[45] and Bohrer,[9]) in which the affected pleural cavities were found to be obliterated by fibrous adhesions, but apparently no systematic study of a series of such necropsies has ever been made.

Heuer[64] not only regards the obliteration of the empyema cavity an example of wound healing, but has plotted curves based upon estimations of the volume of the cavity during the process of healing and concludes that the curve of healing determined by Carrel and DuNuoy for surface wounds is also applicable to the healing of empyema cavities.

The microscopic changes occurring in the development and healing of empyema do not appear to differ fundamentally from those of suppurative inflammation elsewhere. Observations on experimental empyema by Coenen,[25] and by Gay and his associates, and more general observations on inflammation or studies of tissue cultures by Silverberg,[119] Zadek,[139] and Maximow,[88] provide us with much information concerning the cytology of the exudate. Presumably less attention has been paid to changes occurring in the pleura in the later stages when healing is taking place.

Empyema is characterized early by a vascular reaction and the formation of an exudate in which polymorphonuclear leucocytes predominate but which also includes serum, fibrin, and erythrocytes. The granular leucocytes have no power of reproduction, and, as they are acted upon by proteolytic ferments, present in the exudate or in the leucocytes themselves, they soon degenerate. Monocytes, lymphocytes, and histiogenic macrophages also enter into the formation of the exudate and eventually become morphologically indistinguishable from one another.

Gay, Clark and Linton observed in experimental empyema that on the third day only a few polymorphonuclear leucocytes remained. The infiltration of the pleura by polymorphonuclear leucocytes had been replaced by an intensified thickening due to mononuclear cells. The most prominent reaction occurred in the parietal pleura, which at this stage was five times the normal thickness. The serosal layer was intact when the exudate was produced by aleuronat or gum acacia broth injections, but in fatal streptococcic infections fragmentation and necrosis of this layer were observed.

Fibroblasts, according to Coenen, may first be observed at the end of forty-eight hours. They continue to grow through the exudate, but in empyema, as a result of the injection of aleuronat, there was eventually a complete restitution of the pleura without adhesions. On the other hand, MacCallum states that slight inflammatory processes in the pleura

may heal completely, leaving no trace behind, but more severe alterations with fibrinous exudate cause the formation of granulation tissue to replace the exudate, finally binding together the pleural surfaces with adhesions.

THE PROBLEM

From the above discussion of reexpansion of the lung and healing of the empyema cavity, it is apparent that there are a number of questions deserving of further consideration. Is adhesion of the opposed pleural surfaces an important factor in reexpansion of the lung? Does the increased intrapulmonary pressure developed as a result of cough or forced expiration lead to a progressive expansion of the lung? Is a negative intrathoracic pressure necessary or desirable? What magnitude of differential pressure is required to expand a lung restrained by a thickened visceral pleura? How does healing of the empyema cavity occur? What is the condition of the pleura perpheral to the area of encapsulation? Can an empyema cavity be considered healed before complete obliteration of the cavity has occurred? Does restitution of the pleura occur after the healing of empyema?

These questions refer to the manner in which the lung expands and the process by which the cavity heals. A further question refers directly to the application of methods of expanding the lung in the treatment of empyema. *If adequate drainage is provided, is it necessary to employ any method to insure expansion of the lung?* Many surgeons have employed closed drainage, "flapper valves," or blowing exercises for this purpose. Others have considered such measures either futile or unnecessary. Bohrer, for example, has stated that, "It is quite evident that the blow bottle, though it has been used for many years, plays but a small part, if any, in causing the lung to expand. The production and maintenance of a normal or increased negative intrapleural pressure is just as barren of results."

It was in the hope of further clarifying the general problem implied in the above questions that the investigation reported here was undertaken.

MATERIAL AND METHODS

Studies were made on rabbits with experimental empyema and also on empyema in man. Purulent pleural effusions were produced in rabbits by injecting different materials into the pleural cavity. After some preliminary work with defibrinated blood and iodized oil, an aleuronat emulsion staphylococcus method was adopted.

The method consisted, first, of inducing a small pneumothorax, and then injecting 10 to 20 c.c. of aleuronat (2 per cent) and starch (3 per cent). About forty-eight hours later, as a rule, the chest was aspirated; and a turbid, often sanguineous, exudate was recovered. The exudate was then reinjected and 0.5 to 1.0 c.c. of a broth culture of *Staphylococcus aureus* was added. In some of the animals the aleuronat emulsion or staphylococcus injections were repeated. This method was usually successful in causing the formation of a cellular exudate.

The observations on normal rabbits and those with experimental empyema dealt with: (1) gross and microscopic changes in the pleura, (2) the consequences of

open pneumothorax, (3) the effect of differential pressure on healing, and (4) measurements of pressures required to expand the lung.

The studies in empyema in man (patients admitted to the University of Minnesota Hospital) were concerned with: (1) observations at necropsy, (2) clinical observations on obliteration of the cavity, (3) the effect of blowing exercises on reexpansion of the lung, (4) the effects of negative pressure drainage, and (5) fluoroscopic and x-ray observations on resolution of the thickened pleura.

THE GROSS PATHOLOGY OF EXPERIMENTAL EMPYEMA

The appearance of empyema produced in rabbits differs in some details from that observed in man, but in the experimental animal it is possible to follow the various stages and make comparisons with the more isolated observations of the disease in man.

Necropsies were performed on 18 rabbits, one to sixty-two days after the injection of the aleuronat emulsion. The lung on the side of the effusion was always atelectatic, often markedly congested, sometimes pneumonic. The contralateral lung often showed areas of patchy atelectasis, or even complete atelectasis of the middle or accessory lobe. Ultimately, after absorption or removal of the fluid, the lung often attained a stage of apparently normal expansion.

The empyema was usually limited to the side of the chest on which the injection had been made. Sometimes there was to be found, on the contralateral side, a small pleural effusion, clear or bloody, and in the later stages a small amount of thick exudate on the mediastinal pleura, or a few delicate, scattered adhesions.

On the side of the injection, as early as the first twenty-four hours, a turbid or sanguineous fluid was present (differing in appearance from the fluid injected), and a whitish, thick exudate could be observed on the pleural surfaces. By the fifth to tenth day there was usually an abundance of a white, thick, often shaggy exudate not only on the pleurae but sometimes bridging across the pleural cavity. The mediastinum was also thickened by exudate although deviated to the opposite side. A fibrinopurulent pericarditis occurred in two animals. At this stage (five to ten days) the lung was usually loosely adherent to the mediastinum, diaphragm, and sometimes to part of the parietal wall by means of this exudate.

By the sixteenth day and from that time on until the last observation was made on the sixty-second day, the exudate, as a rule, was less fluid and was represented by an encapsulated, white, caseous exudate which appears to be characteristic of inflammation in rabbits. The encapsulation occurred nearly always anteriorly and medially on or close to the diaphragm, and sometimes extended into the fissure between the lower and the middle lobes. Occasionally encapsulation occurred at the base posteriorly, and in one instance multiple encapsulations were observed.

Wherever the visceral and parietal pleurae were permitted to come together as a result of absorption or aspiration of air and exudate, adhesions occurred. As a consequence, the characteristic finding in experimental empyema was an eventual complete obliteration of the pleural cavity by fibrous union except in that area in which the caseous exudate remained. Here no true pleural cavity could be said to remain but merely an abscess with an organizing pyogenic membrane.

There were two exceptions to the rule. One rabbit at the end of eighteen days had about 3 c.c. of fluid encapsulated at the base posteriorly. The remainder of the pleural cavity was free except that it was adherent at several points by delicate string adhesions. The other, at twenty-six days, instead of having universal adhesions had numerous adhesions of various types—sheets, bands, and strings—in addition to an encapsulated cavity which had been drained by open thoracotomy.

The final result of the process is well illustrated by a necropsy performed at the end of sixty-two days. In this instance only about 1 c.c. of thick caseous exudate remained encapsulated in the anterior thorax. There were universal delicate fibrous adhesions limiting the left lung to about 50 per cent of its normal volume. The

chest wall was retracted, the diaphragm elevated, and the mediastinum deviated to that side. The right lung was overdistended, the right pleura entirely normal.

In any consideration of the healing of empyema particular attention must be directed toward that portion of the pleural cavity in which the exudate becomes encapsulated. In this study an attempt was made to observe the healing of the true empyema cavity, but the results were unsatisfactory for the following reasons: (1) rabbits do not tolerate open thoracotomy well even after the exudate has become thick; (2) measurement of the cavity with fluids is inaccurate because of the forcing of fluid out of the chest with forced expiration; (3) introduction of fluid into the cavity sometimes breaks down loose adhesions, increases the size of the cavity, further collapses the lung, and may cause dyspnea, cyanosis, even death; (4) small encapsulated cavities are difficult to locate; (5) the thick caseous exudate is not readily drained from the chest even with irrigating solutions; and (6) roentgenograms do not give accurate information regarding the actual size of the cavity.

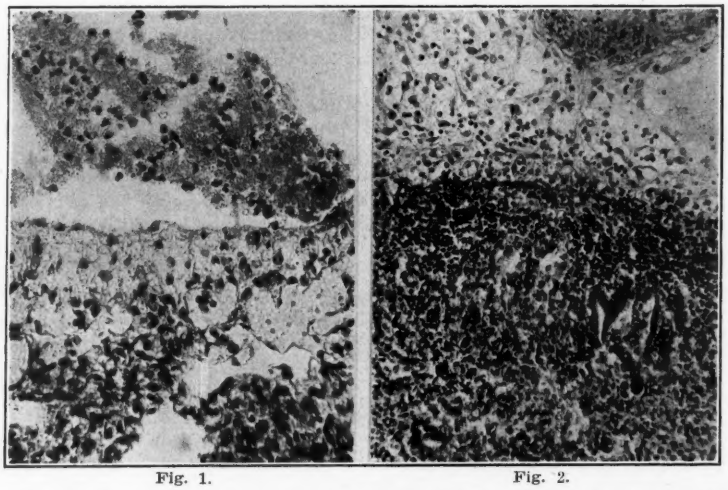


Fig. 1. Fig. 2.

Fig. 1.—Photomicrograph of lung and pleura less than twenty-four hours after intrapleural injection of pneumococcus Type I. There are congestion and edema of the subpleural portion of the lung. Serosal cells are still present. On the surface are exudate cells, chiefly polymorphonuclear leucocytes.

Fig. 2.—Photomicrograph of lung and pleura seven days after intrapleural injection of aleuronat. The lung is atelectatic. Under the pleura may be seen inflammatory cells, chiefly lymphocytes. Tubular structures appearing to be composed of cuboidal epithelial cells may be seen in the peripheral lung parenchyma. The pleura has been replaced by granulation tissue. Fibroblasts and capillaries are clearly demonstrated. Exudate cells are less abundant.

Because of the difficulties attending observations on the healing of the true empyema cavity in rabbits, no systematic study of this process could be made. Fortunately it is this phase of healing that can best be observed in clinical empyema.

MICROSCOPIC OBSERVATIONS

The replacement of the pleural exudate by granulation tissue and the ultimate formation of adhesions between the lung and the chest wall is best observed in microscopic sections.

A study of sections of the lung and pleura eighteen hours after the injection of aleuronat revealed that the lung was markedly atelectatic and congested. The pleural cells at this stage were intact but practically cuboidal in form. On the vis-

ceral surface there was an exudate consisting chiefly of polymorphonuclear leucocytes. A few of these cells could also be seen under the pleura.

A section of the chest wall from the same animal disclosed an infiltration of leucocytes extending deep down into the intermuscular septums. On the surface was a cellular exudate occupying the position of the pleura. No serosal cells were seen.

A section of the lung and visceral pleura twenty-four hours after injection of a culture of pneumococcus Type I was essentially the same as that just described except the lung had been expanded in this experiment and the serosal cells were more flattened (Fig. 1).

Sections obtained from animals four to seven days (Fig. 2) after injection of aleuronat (followed by staphylococcus) revealed that the exudate was less cellular; polymorphonuclear leucocytes were less numerous, but there had been an apparent increase in the number of mononuclear phagocytes. The subpleural lymphatics were dilated and filled with lymphocytes. Serosal cells had disappeared. Numerous fibro-

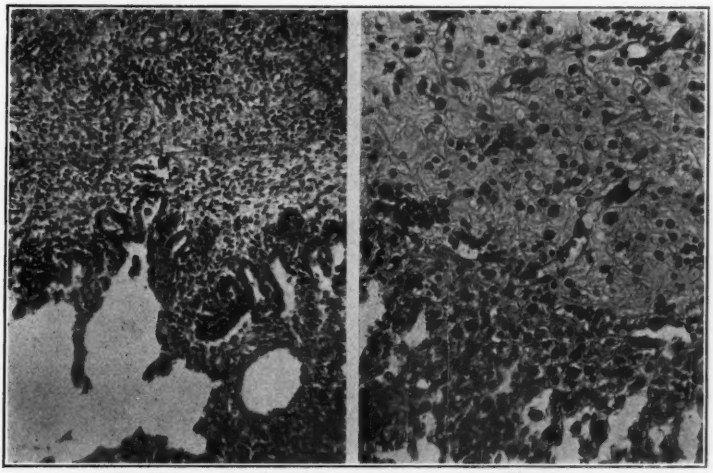


Fig. 3. Fig. 4.

Fig. 3.—Photomicrograph of lung and pleura fourteen days after injection of aleuronat. The atelectatic lung has been partially expanded postmortem. Cuboidal epithelial cells are apparently continuous with cells lining the pulmonary alveoli. Granulation tissue replacing the pleura has become more dense.

Fig. 4.—Photomicrograph of lung and pleura fourteen days after injection of aleuronat. A capillary is shown extending from the lung outward into the pleural granulation tissue.

blasts were present at this stage, and capillaries could be traced from the lung or chest wall extending into the exudate. The formation of granulation tissue was apparently farther advanced on the parietal than on the visceral surface, but this difference was not always convincing.

After fourteen days (Figs. 3 and 4) fewer exudate cells were observed and more connective tissue had formed. A section obtained at the end of twenty-five days demonstrated bands of exudate connecting the lung and chest wall, becoming organized from both the parietal and the visceral extremities (Fig. 5).

In time the exudate was replaced progressively by fibrous connective tissue, which eventually again became more delicate. A specimen obtained at the end of sixty-two days showed the lung adherent to the chest wall by delicate fibrous adhesions with some phagocytes still remaining within the meshes of connective tissue fibrils.

Examination of the slides referred to above revealed a peculiar phenomenon which has, heretofore, received but scant attention. Directly under the visceral pleura, in some of the sections, irregular tubular structures were observed which appeared to be made up of cuboidal epithelial cells (Figs. 2 and 3). Careful study revealed that these tubes communicated with pulmonary alveoli. The cuboidal cells evidently were continuous with the cells lining the alveoli of the lungs. As they were situated at the periphery of the lung, these cells could not represent bronchial epithelium. As they were definitely within the lung parenchyma, they could not be confused with serosal cells. From their morphology and location it is appropriate to regard these cells as derived from alveolar epithelium.

This hyperplasia of alveolar epithelium was observed in specimens four to seven days after injection of aleuronat. It was not a constant feature and when present was usually found only in limited areas of the lung immediately under the pleura.

A specimen was also obtained from a case of empyema in man of about nine weeks' duration which revealed a condition similar to that observed in rabbits

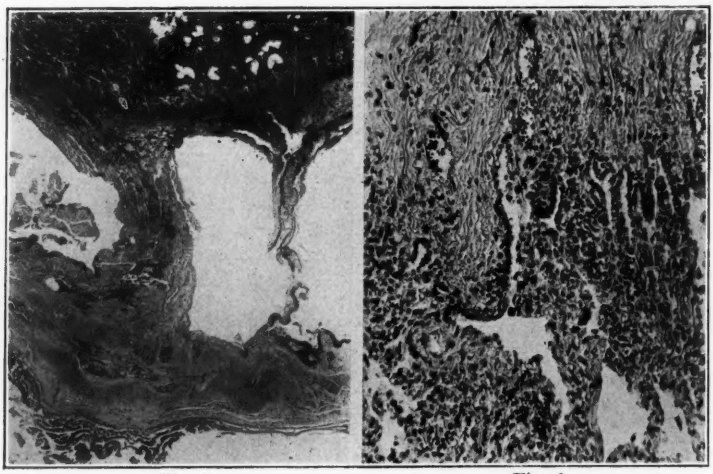


Fig. 5. Fig. 6.

Fig. 5.—Low power photomicrograph twenty-five days after injection of aleuronat, showing lung, chest wall, and intervening exudate. Bands of exudate are becoming organized from both the parietal and the visceral extremities of the bands.

Fig. 6.—Photomicrograph of lung and pleura from a clinical case of empyema of nine weeks' duration. The lung is atelectatic. The visceral pleura has been replaced by dense fibrous connective tissue. Immediately under the fibrous pleura are structures composed of cuboidal epithelium, similar to those observed in experimental empyema.

(Fig. 6). The lung was collapsed, the pleura had been replaced by dense fibrous tissue and immediately under this fibrous layer were structures composed of cells having the appearance of cuboidal epithelium, arranged to form lumina apparently continuous with the collapsed pulmonary alveoli.

OBSERVATIONS ON OPEN PNEUMOTHORAX

Rib resection was performed upon ten rabbits, a tube with an internal diameter of 4 to 5 mm. being inserted into the chest of each. These animals were considered normal, although seven of them had been used for other experiments. Regardless of the anesthetic employed—ether,

novocaine infiltration, or intraperitoneal sodium nembutal—all promptly became dyspneic and cyanotic and all died within a few minutes to ninety minutes after the chest was opened.

Intrapleural pressure measurements varied greatly in different normal rabbits and even from time to time in the same rabbit because of changes in the character of the breathing. These variations before opening the chest extended from –1, –1½ to –4, –4½ cm. of water pressure. After open thoracotomy, measurements obtained by introducing the manometer needle through the thoracotomy opening revealed that the pressure was still slightly negative on inspiration (0, –1 to 0, –2 cm. of water), but even so the lung collapsed. The slightly negative pressure did not prevent cyanosis, dyspnea, and death.

Intrapleural pressure measurements on animals with experimental empyema did not differ materially from those on normal rabbits. These animals, however, often survived the immediate effects of opening the chest. Rib resection was performed under local anesthesia on eight rabbits 7 to 25 days after the introduction of blood, oil, or aleuronat into the pleural cavity. Two died within 30 minutes, one in 6 hours; the others survived 5, 12, 13, 37, and 48 days.

Intrapleural pressures obtained by inserting into the cavity a needle connected with a water manometer varied from +½ to –2 cm. of water. These pressures did not bring about an expansion of the lung.

In order to check the observations of Reineboth, a modification of one of his experiments was carried out on two normal rabbits. The results of these two experiments were practically identical. One is reported here.

A rabbit was anesthetized with ether. The muscles of the thorax were removed down to the ribs and intercostal muscles. An upper abdominal incision was also made. Then, with the room darkened, a small light was inserted through the abdominal incision directly under the central tendon of the diaphragm. With this method of transillumination it was possible to observe the movements of the lung. Open pneumothorax was then obtained by inserting a needle or tubes of various sizes into the chest, with the following results:

1. A 19 gauge needle (internal diameter about 0.75 mm.) was inserted into the chest on the right side. The lung rapidly collapsed to about one-third of its normal volume as a pneumothorax developed. The lung movements, however, continued, following those of the thorax; i.e., inflation on inspiration, deflation on expiration. The heart was displaced to the left.

2. A tube with an internal diameter of 2.5 mm. was inserted. Further collapse of the lung occurred until it appeared to be only about one-fifth of the original volume. Now a paradox motion of the lung was observed on the side of the open pneumothorax. On inspiration the lung receded; on expiration it appeared to increase in volume.

3. A tube with an internal diameter of 5 mm. was inserted. The observations were about the same as with a 2.5 mm. tube.

4. A large opening about 2 by 2.5 cm. was made in the chest wall. The lung movements were approximately the same as with the tubes of 2.5 mm. or 5 mm., but

direct inspection through the large opening revealed that the volume of the lung did not change appreciably, the paradox motion observed being due almost entirely to deflections of the mediastinum with each cycle of respiration, the collapsed lung following the movements of the mediastinum.

Measurements at necropsy revealed that the diameter of the trachea was 5 mm., of the right main bronchus, 3 mm. In general these experiments confirm those of Reineboth, although no observations were made upon the effect of forced expiration.

THE EFFECT OF DIFFERENTIAL PRESSURE AFTER THORACOTOMY

Experiments were performed on four rabbits to determine whether the effects of an open pneumothorax could be overcome by means of a prolonged administration of differential pressure. For this purpose a negative pressure box was constructed. The animal was placed in this box, the body inside, the head, however, protruding through an opening in a piece of rubber dam. When the box was sealed, a negative pressure applied constantly by means of a water suction pump and the degree of negative pressure controlled by means of a valve, a method was provided for continuing a differential pressure for as long a time as desired.

An open thoracotomy was performed before placing the rabbit in the box. As the results of the experiments varied considerably because of unforeseen complications, they will be outlined individually.

Experiment 1, Rabbit 19: A rib resection was performed on a normal rabbit under local anesthesia, a 5 mm. tube being inserted into the pleural cavity. The animal immediately became dyspneic, cyanotic, and listless. Within ten minutes he was placed in the negative pressure box and a pressure of −7 cm. of water applied. Immediately the respirations became easier, cyanosis disappeared, he became brighter and attempted to move although restrained by ear restraints. The following day he was found to be cyanotic and dyspneic again. It was found that negative pressure was no longer being supplied. As soon as this fault was corrected, he immediately recovered from the cyanosis and dyspnea. This rabbit, however, refused food and water and lived only about sixty hours after the thoracotomy. The cause of death was not determined. The lungs at necropsy were completely expanded and of normal color. The visceral and parietal pleurae were approximated but could be readily separated. There was no gross exudate. The tube had come out of the chest.

Experiment 2, Rabbit 20: Open thoracotomy was performed under local anesthesia, and 1 c.c. of a culture of pneumococcus Type I was injected into the pleural cavity. Dyspnea and cyanosis appeared. He was placed in the negative pressure box, a pressure of −4.5 cm. of water being maintained. Dyspnea and cyanosis promptly disappeared, but the animal lived only about eighteen hours. At necropsy its lungs were fully expanded. The pleura grossly showed little change but on microscopic examination showed a small amount of a cellular exudate, chiefly polymorphonuclear leucocytes. As rabbits are susceptible to pneumococcus, usually developing a septicemia, this organism was not used in any further experiments.

Experiment 3, Rabbit 21: Open thoracotomy was performed and 10 c.c. of an emulsion of aleuronat was injected into the pleural cavity. The animal immediately became cyanotic and dyspneic but was relieved when placed in the box with a pressure of −5 cm. of water. Three hours later this rabbit broke the ear restraints and pulled his head into the box. When removed from the box he appeared normal,

showing no signs of cyanosis or dyspnea. On inspection the inflated lung could be seen sealing the opening in the chest. The lung was touched lightly with a hemostat, and it immediately collapsed. The rabbit once again became cyanotic and dyspneic. This condition was again corrected by replacing him in the box. Unfortunately the box was upset during the night by a dog and the rabbit died with an open pneumothorax.

Experiment 4, Rabbit 27: An emulsion of aleuronat (15 c.c.) was injected into the left pleural cavity to stimulate the formation of an exudate. Two days later an open thoracotomy was performed. A thin turbid fluid was present in the pleural cavity. A 4 mm. tube was inserted into the chest. The rabbit appeared dyspneic and cyanotic. He was placed in the negative pressure box, a pressure of −4 to −7 cm. of water being maintained. The cyanosis and dyspnea immediately disappeared.

He remained in the negative pressure box for forty-eight hours, then was removed and returned to a cage. Respiration was apparently normal. The tube had been pushed out of the chest. The lung covered by an exudate sealed the thoracic opening.

The rabbit refused food and water and died the following day. He, therefore, lived five days after the injection of aleuronat, three days after opening the chest, one day after removal from the negative pressure chamber.

At necropsy the right lung and pleura were normal. The left lung was expanded and adherent loosely to the parietal pleura by means of a layer of exudate. There was no free fluid. The pericardium contained a white, thick, pasty exudate. Microscopic sections showed beginning organization of the exudate between the lung and chest wall.

These four experiments show clearly that the effects of open pneumothorax can be overcome by means of a continued differential pressure. If the lung is kept in contact with the parietal pleura for a sufficiently long period of time in the presence of pleural inflammation, the opposed surfaces adhere and the lung remains expanded even after the differential pressure is discontinued. However, even though the negative pressure may prevent the animal from dying of open pneumothorax, the mortality under the conditions of these experiments remains high, all four of the rabbits having died of one cause or another.

OBSERVATIONS ON HEALING AFTER REEXPANSION OF THE LUNG AND CLOSURE OF THE WOUND

Experiments were carried out on four rabbits in which attempts were made to expand the lung by means of positive pressure in the presence of an intercostal thoracotomy performed six to eight days after the injection of aleuronat emulsion, then to suture the wound tightly and observe the effect of reexpansion of the lung on healing at the end of two to nine days.

The results were not entirely satisfactory because pneumothorax was not completely excluded, because fluid re-formed, or because the thoracic wound became infected. One to 15 c.c. of fluid or air was found in the chest at necropsy in each rabbit, the remainder of the pleural cavity being obliterated by a layer of thick exudate. In spite of the failure to attain complete expansion of the lung, the experiments indicate that the pleural cavity is more or less completely obliterated depending upon the completeness of approximation of the lung and thoracic wall. It is not necessary that adhesions form at one point and then spread out to involve the remainder of the pleural cavity by a slow process of gradual extension.

REEXPANSION PRESSURE MEASUREMENTS

One of the important questions involved in a consideration of reexpansion of the lung is the amount of pressure required to bring about reexpansion. Obviously the thickened pleura, pulmonary atelectasis, and perhaps other factors may greatly alter the differential pressure required to inflate the lung. In order to measure this pressure, the lungs of rabbits with experimental empyema were inflated either after the lungs had been removed or with the lungs in situ.

Lungs removed from the body of the animal could be inflated either by applying positive pressure to the trachea or by placing them in a jar to which negative pressure was applied, the trachea being exposed to the atmospheric pressure. With the lungs in situ, the anterior half of the chest having been removed, inflation could best be accomplished by means of positive pressure. The technic of these experiments is so simple as to require no detailed description. Positive pressure was obtained by means of compressed air, negative pressure by means of a water suction pump. The degree of pressure was controlled by a water or mercury positive or negative pressure valve and measured with a water manometer. The experiments are detailed below. The pressures recorded were continued for about five minutes.

Experiment 1, Rabbit N-2. Empyema, left—five days. (Aleuronat-staphylococcus.)

Gross examination: Right lung and pleura normal. Left pleural cavity contained air and about 15 c.c. of turbid, sanguineous fluid. The left middle lobe was atelectatic and was covered with a thick greenish exudate.

POSITIVE PRESSURES (WITH LUNGS REMOVED FROM THE BODY)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Normal expansion	Middle lobe atelectatic. Normal expansion of rest of lung.
+27 cm. water		Expansion of the middle lobe occurred only when this pressure was reached.

Experiment 2, Rabbit N-3. Empyema, right—seven days.

Gross examination: Left lung and pleura normal. Right—thick exudate over midportion of lung anteriorly and posteriorly. The pleural cavity was obliterated. There was no free fluid.

NEGATIVE PRESSURES (WITH LUNGS REMOVED)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
−12 cm. water	Expansion of apex only.	Fair expansion. Atelectatic areas remain.
−17 cm. water	Apex, anterior border, and base of lung are overdistended. Much of lower and middle lobes are atelectatic.	Lung overdistended. A few atelectatic patches remain.
−23 cm. water	Two small areas covered by exudate do not reexpand. Rest of lung overdistended.	Overdistended. No atelectasis.

Experiment 3, Rabbit N-1. Empyema, right—fourteen days.

Gross examination: Left—3 c.c. of thin, turbid fluid in pleural cavity. A few areas of patchy atelectasis in lung. Right—40 c.c. of thin, turbid fluid in pleural cavity. Right middle lobe is atelectatic and is covered with a thick exudate.

NEGATIVE AND POSITIVE PRESSURES (LUNGS REMOVED FROM CHEST)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
−11 cm. water	Beginning expansion of upper lobe.	Beginning expansion.
−14 cm. water	Incomplete expansion.	Good expansion. A few atelectatic areas remain.
−16 cm. water	Middle lobe atelectatic. Rest of lung expanded.	Small atelectatic areas remain.
+25 cm. water	Middle lobe atelectatic.	Complete expansion. Overdistention.
+43 cm. water	Middle lobe expanded in about three minutes.	

Experiment 4, Rabbit 45. Empyema, left—nineteen days.

Gross examination: Right lung and pleura practically normal. Left—a cavity of 2 to 3 c.c. had been drained posteriorly. The lung was atelectatic and bound down by adhesions.

POSITIVE PRESSURES (LUNGS REMOVED FROM BODY)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+10 cm. water	Normal inflation.	Incomplete expansion.
+33 cm. water		Lung inflated except at wall of cavity. This lung at +33 cm. water pressure had less than one-half the volume of the other at +10 cm. water pressure.

Experiment 5, Rabbit N-14. Empyema, left—seven days.

Gross examination: Right lung and pleura normal. Left pleural cavity completely obliterated by adhesions except anteriorly where there were 1 to 2 c.c. of thick, inspissated, greenish exudate.

POSITIVE PRESSURES (LUNGS IN SITU)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+10 cm. water	Practically normal expansion.	Less than normal.
+15 cm. water	Slight overdistention.	Less than normal.
+40 cm. water		Prevented from normal expansion by adhesions.
+15 cm. water After removal of lung		Good expansion.

Experiment 6, Rabbit N-16. Empyema, left side—fourteen days.

Gross examination: Right pleura normal. Some atelectasis of right lower lobe. Left—cavity obliterated by shaggy exudate. There are also about 5 c.c. of thin, reddish fluid encapsulated at base.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Fair expansion. Atelectatic areas remain.	Not expanded.
+18 cm. water	Well expanded, except for atelectasis of lower lobe.	Not expanded.
+25 cm. water	Complete expansion in about three minutes.	Lung fills pleural space fairly well.
+45 cm. water		Leakage occurred. Lower lobe incompletely expanded.

Experiment 7, Rabbit N-6. Empyema, left—sixteen days.

Gross examination: Right lung and pleura normal. Left—about 2 c.c. of thick exudate. Rest of cavity obliterated by adhesions.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Fair expansion.	Incomplete expansion.
+18 cm. water	Overdistended. Some atelectasis remains.	Good expansion. Lung fills pleural cavity.
+23 cm. water	All atelectatic areas disappear. Overdistended.	Practically complete expansion. Smaller than opposite lung.

Experiment 8, Rabbit N-18. Empyema, left—eighteen days.

Gross examination: Right side normal. Left—about 2 c.c. of thick, caseous, encapsulated exudate anteriorly. Cavity obliterated elsewhere.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Fair expansion. Atelectasis in middle lobe.	Incomplete expansion.
+22 cm. water	Atelectatic areas disappear in five minutes. Lung overdistended.	Incomplete expansion.
+45 cm. water		Lung fills pleural cavity but there is atelectasis in region of exudate. No overdistention.

Experiment 9, Rabbit N-15. Empyema, right side—twenty-eight days.

Gross examination: Left side—normal lung and pleura. Right side—complete obliteration of pleural space by very delicate adhesions except for 1 to 2 c.c. of thick exudate anteriorly extending between upper and middle lobes. Lung appears normal.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Beginning expansion.	Beginning expansion.
+15 cm. water	Normal except in region of exudate.	Normal expansion.
+18 cm. water	No increase.	Overdistention.
+45 cm. water	Good expansion. Adhesions prevent overdistention.	

Experiment 10, Rabbit N-17. Empyema, right side—thirty-three days.

Gross examination: Left side—normal. Right—about 2 to 3 c.c. of thick exudate anteriorly, interlobar, and between lung and diaphragm. Universal delicate adhesions obliterating pleural cavity.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Less than on other side.	Incomplete expansion.
+14 cm. water	Incomplete expansion.	Normal expansion except for atelectatic areas.
+18 cm. water	Incomplete expansion.	All atelectatic areas disappear. Lung overdistended.
+25 cm. water	Lower and accessory lobes are overdistended. No increased expansion where fixed anteriorly.	
+60 cm. water	Overdistended but fixed by adhesions anteriorly.	

Experiment 11, Rabbit N-10. Empyema, left—sixty-two days.

Gross examination: Right—lung overexpanded. Mediastinum deviated to left. Right pleura normal. Left—lung about half of normal volume, bound to chest wall by universal adhesions. About 1 c.c. of caseous exudate at base posteriorly.

Positive Pressures (Lungs in Situ)

PRESSURES	INFLATION OF RIGHT LUNG	INFLATION OF LEFT LUNG
+12 cm. water	Beginning expansion.	No expansion.
+18 cm. water	Good expansion. Some atelectatic areas.	Anterior portion of lung shows some expansion.
+25 cm. water	All atelectatic areas disappear. Lung overexpanded.	Some increase of expansion.
+60 cm. water		Lung failed to expand to normal volume. Rupture of alveoli occurred and air escaped, separating pleural adhesions.

These experiments demonstrate convincingly that the differential pressure required to expand the lung on the side of an empyema is significantly greater than the pressure required to expand a normal lung or an atelectatic lung with a normal pleura. Pleural pressure measurements recorded before and after removal of the lung reveal that it is chiefly the pleural exudate or fibrous adhesions rather than an intrapulmonary factor which interferes with expansion of the lung.

The pressures in these experiments were uninterrupted and continued as a rule for five minutes. It is quite probable that a differential pressure altered by respiratory movements and continued over a long period of time would be more successful in bringing about an expansion of the lung. It is clear, however, that the positive intrapulmonary pressure developed in normal respiration is not great enough to overcome the resistance of the thickened pleura. It is also apparent that if the lung is to be expanded by differential pressure, it is best done before complete fibrosis of the pleura has occurred. After fibrous adhesions have formed to bind down the lung, it is possible to apply a differential pressure which will tear the lung without expanding it to a normal volume.

SUMMARY OF ANIMAL EXPERIMENTS

Encapsulation of the exudate in rabbits occurs as a rule anteriorly on or near the diaphragm. The remainder of the pleural cavity is usually obliterated by fibrous adhesions.

Microscopic sections reveal the replacement of the visceral and parietal pleurae by granulation tissue and the formation of fibrous adhesions between approximated pleural surfaces. These adhesions in the course of several weeks become more delicate but were not observed to disappear.

During the acute stage hyperplasia of subpleural alveolar epithelium may be observed.

After the chest is opened, the lung collapses; and although the intrathoracic pressure may be slightly negative on inspiration, the lung fails to expand either on inspiration or expiration. However, if the opening in the chest is sufficiently small, the lung remains in a position of partial collapse, and movements of the lung follow those of the thorax, expansion occurring on inspiration.

If, after the chest is opened in the presence of a pleural exudate, the lung is maintained in a position of complete expansion by means of *prolonged* differential pressure until adhesions have formed, the lung will remain permanently expanded after the differential pressure is discontinued.

The amount of pressure required to expand a collapsed lung restrained by a pleural exudate depends primarily upon the resistance offered by the thickened pleura. When the lung is fixed by fibrous adhesions, alveoli may be ruptured by a pressure which fails to expand the lung.

OBSERVATIONS ON CLINICAL EMPYEMA

No evidence need be added to that already available to prove that the process of encapsulation of the pleural exudate occurs in empyema in man, as in experimental animals, by adhesion of opposed pleural surfaces. Roentgen films after injection of air or lipiodol into the chest, direct observation of the empyema cavity through a thoracotomy opening, and the occurrence of multiple encapsulations afford conclu-

sive evidence that encapsulation does occur, usually posteriorly, and that it occurs regardless of the method of drainage, indeed, in many instances before aspiration or any other form of drainage is attempted.

Evidence concerning the nature of changes occurring in the pleura peripheral to the zone of encapsulation is less abundant. Since I have become interested in the problem, there have been opportunities to observe the necropsies on two patients dying with empyema. One of these was a man, aged sixty-two years, who had an aortic aneurysm complicated by a pneumococcic empyema of about nine weeks' duration. There was a well-encapsulated cavity posteriorly having a capacity of about 100 to 200 c.c. The visceral pleura in this region was about 3 to 4 mm. in thickness, the parietal pleura about 1 cm., and the two were intimately adherent at the periphery of the cavity. The remainder of the pleural cavity was not completely obliterated, but there were many sheets and bands of adhesions between the two layers. In a number of places, particularly anteriorly, there were areas in which the opposed surfaces appeared normal.

The other necropsy was performed on a man, aged twenty-four years, who had had chronic empyema and amyloidosis. In this instance there was a large cavity occupying about two-thirds of the hemithorax. The pleurae were much thickened, particularly the parietal pleura which was 1 to 2 cm. in thickness. The collapsed lung was bound intimately to the mediastium, diaphragm, and a portion of the chest wall, the pleural cavity in this region being completely obliterated by fibrous adhesions.

The healing of the empyema cavity itself in the presence of either closed or open drainage is unquestionably one of actual obliteration by union of the opposed, inflamed pleurae. The progress of this process of obliteration has been followed by measurements of the volume of the cavity from time to time and by roentgen examinations after the injection of lipiodol into the cavity (Fig. 7 *F*). Both of these methods reveal that the cavity actually becomes smaller by progression of the angle of fusion until finally complete obliteration and healing have occurred.

An attempt was made to obtain data similar to those reported by Heuer[64] on the healing of empyema cavities. Curves based upon cavity measurements gleaned from the University of Minnesota Hospital records had been plotted already by Bowers.[11] Some of the individual curves were similar to Heuer's, but others were so irregular as to make analysis impossible. In some instances cavity measurements indicated that the cavity temporarily increased in size. These routine volume estimations are subject to errors due to displacement of fluid by coughing, distention of large cavities by the weight of the fluid, incomplete filling due to faulty posture, etc., and apparently are not sufficiently accurate to be relied upon. No effort is made therefore to adapt a mathematical formula to the curve of healing.

THE EFFECT OF BLOWING EXERCISES

Observations under the fluoroscope on the effects of blowing exercises on the lung in the presence of an intercostal closed water seal drainage

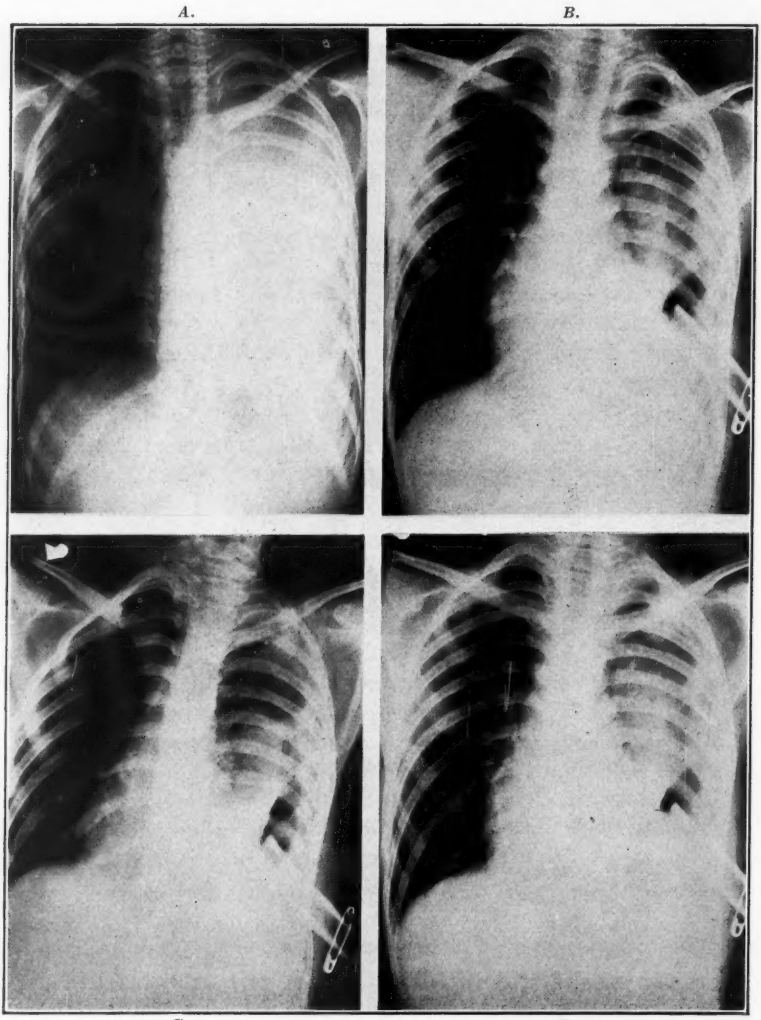


Fig. 7.—*A*, Roentgen film of chest in a case of postpneumonic pneumococcus empyema after treatment by repeated aspirations over a period of forty days. There is a diffuse density on the left.

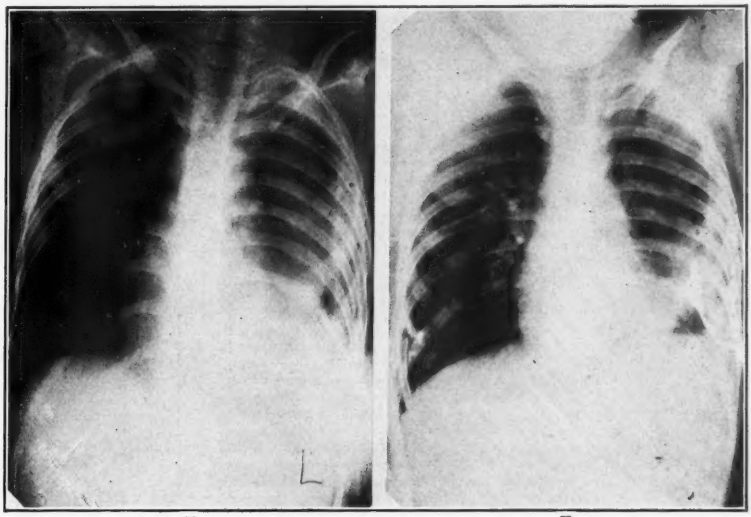
B, Film of chest of same patient two days after rib resection and drainage, showing an encapsulated empyema cavity with thickened pleura. Drainage was complete.

C, Film of chest taken immediately after 7-*B* with patient attempting to reexpand the lung by means of a maximal blowing effort (blow bottle). There is partial expansion of the lung.

D, Film of chest taken shortly after 7-*C*. Normal respiration has been resumed. The lung has returned to a volume about equal to that before blowing.

system revealed that partial expansion of the lung occurred as a result of forced expiration. When the drainage tube, however, was disconnected, an open drainage being substituted for the closed system, the expansion of the lung which had been gained was no longer maintained.

In order to obtain further information, roentgen films of the chest were made before, during, and after the employment of blowing exercises in three children. Two of these failed to blow hard enough to influence the size of the empyema cavity (Fig. 11 *B*). The other was able to reduce the size of his cavity during the act of blowing (Fig. 7 *B, C, D*) but films taken immediately afterward showed no appreciable increase in the expansion of the lung. When a continuous differential pressure



E. *F.*

Fig. 7.—*E*, Film of chest after negative pressure drainage had been continued for three days. The lung is expanding to the chest wall. Note the rubber sponge employed to seal the wound.

F, Lipiodol filling to show residual cavity twenty-four days after rib resection.

was provided in the same case, the lung remained expanded and the empyema cavity was markedly reduced in size (Fig. 7 *E*).

OBSERVATIONS ON NEGATIVE PRESSURE DRAINAGE

Thirteen patients with postpneumonic pneumococcic empyema were treated by a method which combined rib resection with negative pressure drainage. After resection of a rib by the usual technic, a rubber and fabric tube with an internal diameter of about 12 mm. was inserted into the chest. A long tube (25 cm. or longer) was used to permit connection with the negative pressure system.

In some instances observations were made on open drainage for a few days. Then a continuous differential pressure was provided by means

of negative pressure applied to the drainage tube. In some preliminary observations a system of bottles was employed, but more constant differential pressure was obtained by a system similar to that described by Perthes[99] in 1898. It consists of a water suction pump, a pus trap, a negative pressure mercury valve to control the maximum pressure, and a manometer to record the pressure and the fluctuations due to respiration.

An original method was employed to seal the wound. The tube was passed through a small opening in a rubber sponge. The sponge was smeared with a generous coat of vaseline, fitted tightly against the chest wall, and held in place by means of adhesive tape and an elastic bandage. A safety pin through the tube, between the sponge and the chest

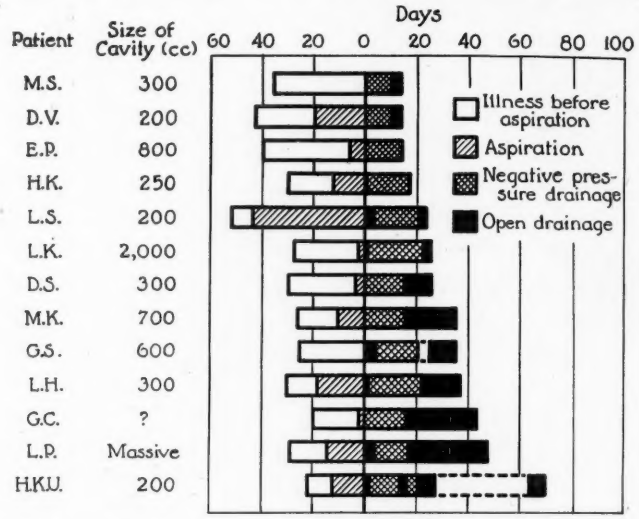


Fig. 8.—Graph to show different phases of the treatment of thirteen cases of pneumococcic empyema in which negative pressure was employed after rib resection. The period prior to the use of continuous drainage is shown to the left of the vertical line. Two patients had two separate cavities drained (G. S. and H. K. U.). The records of three patients are excluded because the negative pressure was interrupted rather than continuous. The periods of drainage in these patients were thirty-eight, forty-seven, and fifty-eight days. In two other patients continuous negative pressure was begun but abandoned in a few days because the patients had multiple encapsulations. These multiple pockets were apparent on the original films.

wall, kept the tube in place, and the skin edges were protected by vaseline strips.

With the large drainage tube and the controlled negative pressure, complete drainage was provided and the lung quickly reexpanded. The pressure varied from –10 cm. to –30 cm. of water beginning with the lower figure and increasing as desired. Sometimes the drainage tube became plugged and had to be washed out or even removed for cleaning, but the employment of Dakin's irrigations either through a T tube or by means of a small catheter inserted into the cavity beside the drainage tube usually sufficed to maintain the patency of the drain.

The duration of illness (including pneumonia) prior to surgical drainage and the period of drainage of thirteen patients treated in this manner has been plotted (Fig. 8) and compared with the period of drainage in twelve patients who were treated by the usual methods of closed or open drainage (Fig. 9). All the patients had postpneumonic pneumococcic empyema. The period of drainage is selected rather than the total period of healing because before healing was complete many of the patients were dismissed to be further treated by the local physician. The time required for closure of the sinus is, therefore, not accurately known. The total period of drainage refers to the interval after open or closed drainage was instituted until the cavity was approximately the size of the drainage tube, or measured 10 c.c. or less, at which time the tube was removed.

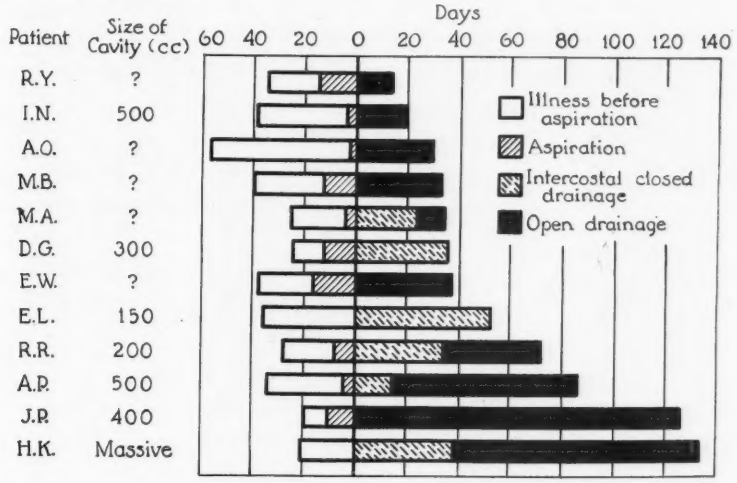


Fig. 9.—Graph to show different phases of the treatment of twelve cases of pneumococcic empyema. The usual methods of closed or open drainage were employed.

The results although concerned with only a small number of cases suggest that a combination of adequate drainage and a satisfactory application of differential pressure results in a rapid decrease in the size of the empyema cavity as a consequence of approximation and adherence of the granulating pleural surfaces.

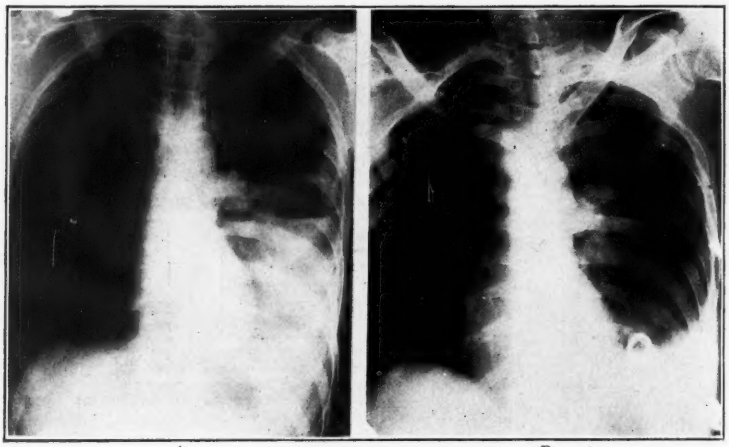
More convincing evidence is to be found in the serial roentgenograms of individual cases. Examination of films (compare Figs. 7, 10, 11) reveals that the lung promptly reexpands and remains in a position of reexpansion when drainage is complete and a satisfactory differential pressure is employed continuously. The completeness of approximation of the visceral and parietal pleurae varies undoubtedly with the size of the original cavity, the thickness and stage of inflammation or healing

of the pleura, and the efficiency with which the drainage and the differential pressure are maintained. These factors, to a large extent, determine the rate of obliteration of the cavity.

Negative pressure drainage was also employed in five cases of chronic empyema. Two had bronchial fistulas. Three showed some degree of expansion, but in only one did expansion appear to be complete. Expansion of the lung, however, did not hasten healing of the cavities.

RESOLUTION OF THE THICKENED PLEURA

An attempt was made to determine what happens to the thickened pleura after healing of an empyema cavity. Fluoroscopic examination and serial roentgen films on ten patients treated by different methods revealed that the pleura gradually became thinned out over a period of



A. *B.*

Fig. 10.—*A*, Film showing postpneumonic pneumococcic empyema. Encapsulated cavity contained air and 800 c.c. pus.

B, Film taken after three days of drainage combining rib resection and negative pressure. No cavity can be demonstrated. Drainage was continued for fourteen days to insure complete healing. The cavity measured 5 c.c. after removal of the tube.

months and that the diaphragm, which was usually elevated and more or less fixed, in time enjoyed greater freedom of movement. In a patient followed for one year after complete healing of an empyema cavity there was still, however, some thickening of the pleura and slight limitation of motion of the diaphragm due to adhesions in the costophrenic angle (Fig. 12 *B*).

A lobectomy performed for bronchiectasis upon a patient who had had empyema more than four years earlier revealed that the pleural cavity, so far as it was explored, was completely obliterated by delicate fibrous adhesions. This condition was similar to the end stage observed in empyema in rabbits.

SUMMARY OF CLINICAL OBSERVATIONS

The gross and microscopic pathology of clinical empyema is fundamentally the same as in experimental empyema so far as observations have been made. The empyema cavity becomes obliterated by the progressive formation of fibrous adhesions between the parietal and the visceral pleurae.

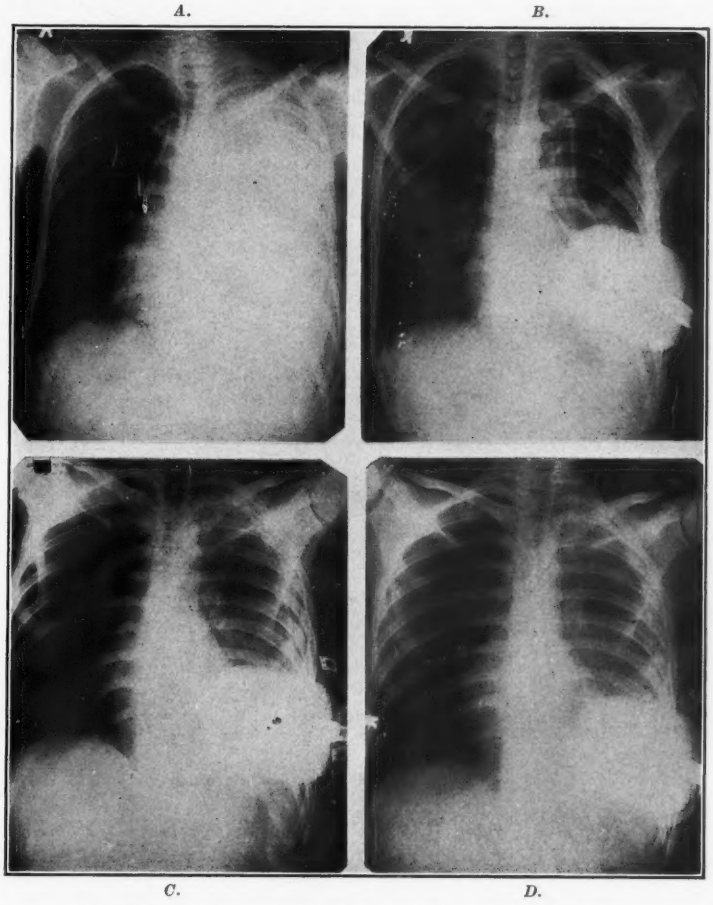


Fig. 11.—*A*, Film showing massive postpneumonic pneumococcic empyema twenty-six days after onset of pneumonia.

B, Film of same patient two days after rib resection and open drainage. At operation 2000 c.c. pus were removed. Mediastinum is stable but the left lung is partially collapsed, a large open pneumothorax being present. This film was taken while the patient was making a maximum blowing effort. The measured blowing pressure was only 20 cm. of water. No expansion of the lung was observed in comparing films taken before, during, and after the blowing exercises.

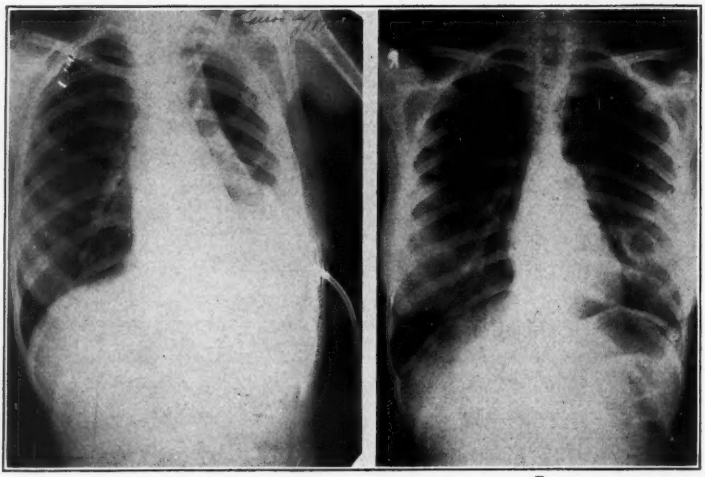
C, Film after seven days of continuous negative pressure drainage. The lung is expanded, only a small pneumothorax remaining. The visceral and parietal pleurae are both thickened, especially the latter.

D, After fourteen days of negative pressure drainage there is practically complete expansion of the lung. Negative pressure was continued for a total period of twenty-one days. The tube was removed two days later.

In the presence of a true open thoracotomy the transient expansion due to forced expiration (blowing exercises) does not appear to be maintained.

The combination of a large drainage tube and continuous negative pressure drainage led to practically complete expansion of the lung and rapid healing in thirteen cases of acute empyema but was unsuccessful in chronic empyema.

The thickened pleura of acute empyema gradually becomes thinned out, but as late as one year after healing there may remain some thickening of the pleura and tenting of the diaphragm as revealed by fluoroscopy and roentgen films.



A. *B.*

Fig. 12.—*A*, Film of chest after five days of closed intercostal drainage, employing the water seal method. Satisfactory expansion of the lung was not obtained because of failure to keep the system closed while the pus trap was being emptied. Even after correction of this error complete expansion did not occur. Eventually rib resection was performed for open drainage. Healing required more than three months.

B, Film of same patient one year after healing of cavity. Fluoroscopic examination revealed some tenting and limited motion of the left leaf of the diaphragm. Films reveal some thickness of the pleura and adhesions in the costophrenic angle.

DISCUSSION

The experimental and clinical observations reported here considered in connection with those previously reported in the literature permit us to formulate definite ideas regarding the process of reexpansion of the lung under the various forms of clinical management.

When a true open drainage is employed, the collapsed lung does not exhibit a natural tendency to expand. On the contrary, immediately after the chest is opened, the lung remains collapsed because of its own elasticity and because of the resistance to expansion offered by the thickened visceral pleura. The degree of collapse is determined, largely, by

the extent of adhesions which have formed between the pleural surfaces prior to opening the chest. The expansion of the lung so frequently alleged to occur immediately after withdrawal of pus is best explained on the basis of release of tension in the presence of an encapsulated empyema cavity. After the readjustment due to the changes in intrathoracic pressure the lung shows no further immediate tendency to expand (Figs. 11 *B* and 13).

Three ideas have been offered in explanation of the subsequent expansion which takes place: (1) negative intrathoracic pressure, (2) an increased intrapulmonary pressure, and (3) contraction of granulation tissue at the angle of fusion of the visceral and parietal pleurae with progression of this angle of fusion in the process of healing.

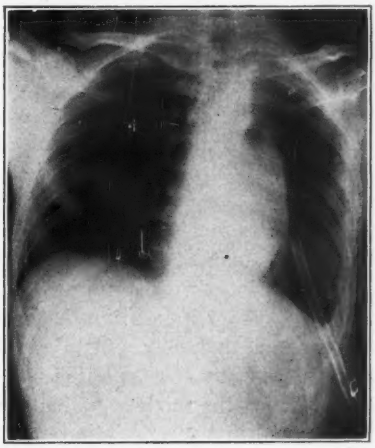


Fig. 13.—Film of chest of patient referred to hospital four weeks after rib resection and open drainage for a postpneumonic empyema with thick pus (organism undetermined). This case illustrates the error of too early open drainage. The mediastinum was stable, but there was nearly complete collapse of the lung. Treated by open drainage, healing required about one hundred and twenty days.

In the experiments with open pneumothorax reported here, the slightly negative intrathoracic pressure created in normal respiratory movements was inadequate to expand even a normal lung. It is obviously inadequate to expand the lung against the resistence offered by a thickened pleura. It is undoubtedly true that a pus-soaked dressing overlying a drainage tube may act as a valve and aid in building up a negative intrathoracic pressure, but such a mechanism is intermittent and unreliable.

The increased intrapulmonary pressure occurring in the expiratory phase of normal respiration has never been demonstrated to be a significant factor in reexpansion. Forced expiration or cough unquestionably expands the lung momentarily, but the expansion gained is imme-

diately lost in the succeeding inspiration. With an open thoracotomy there is no mechanism for maintaining each increment of expansion gained in this manner. Theoretically a fibrinous exudate is capable of holding the lung in contact with the chest wall, but x-ray films before and after blowing exercises fail to reveal any decrease in the volume of the cavity. Although it is not denied that blowing exercises may be beneficial by inflating atelectatic areas, or by exercising muscles of respiration, no convincing evidence has so far been advanced in support of the idea that a transient increase in the intrapulmonary pressure leads to a permanent increase in the volume of the lung.

The third explanation, expansion of the lung by progression of the angle of fusion in the process of healing, is supported by ample experimental and clinical evidence.

When some form of continuous negative pressure is employed, the lung expands by virtue of the differential pressure created. The degree of expansion attained depends primarily on the relationship between the amount of pressure used and the resistance offered by the elastic lung and the thickened visceral pleura.

If expansion is sufficiently complete to permit of close approximation of all or part of the opposed pleural surfaces, fusion of the surfaces by adhesions is possible and the lung may remain in the expanded position after differential pressure is discontinued. Alexander[2] makes use of this process in the first stage of his two-stage lobectomy when he produces adhesions by abrading the pleura with gauze.

Theoretically it is possible that if infection is severe at the time the pleurae are brought together, they may fail to adhere or encapsulated effusions may develop. Secondary encapsulation requiring drainage occurred in two instances among the thirteen patients treated by negative pressure drainage, but as multiple encapsulations may occur under other forms of treatment or before treatment is instituted, the development of these secondary encapsulations cannot be regarded as a serious objection to the method.

Expansion of the lung when aspiration is the sole method of treatment must be identical with the mechanism described for negative pressure drainage except that in this instance the drainage is intermittent and fluid may re-form during the interval.

Management by aspiration and air replacement requires special mention. The theory of Danna[31] that absorption of air from a closed pneumothorax aids in expansion appears reasonable, as this process is so well known in the reexpansion of the lung following artificial pneumothorax. No data, however, are available on the rate of absorption of gases when the normal pleura is replaced by granulation tissue. Clinical observations indicate that absorption of gases does take place even under these conditions, although in some instances a large closed pneumothorax has appeared to delay rather than hasten expansion.

Experiences with pyopneumothorax in rabbits reveal that as a rule the pleural space eventually becomes obliterated by adhesions even in the presence of a closed pneumothorax.

The differential pressure required for reexpanding the lung exceeds the normal intrathoracic pressure because it is necessary to overcome not only compression atelectasis but also the resistance offered by a thickened pleura. In some instances, no doubt, pneumonia or pulmonary fibrosis may further interfere with expansion.

Observations on experimental empyema demonstrate that differential pressures of 10 to 60 cm. of water continued for about five minutes may or may not be adequate to bring about a satisfactory expansion of the lung. Pressures of –10 to –30 cm. of water continued over a period of several days usually sufficed in clinical cases of empyema. Perthes[99] who formerly used negative pressure greater than –100 cm. of water now regards these pressures as excessive and no longer exceeds –30 cm. of water pressure in acute cases. Storch[125] stated in 1890 that pressures of more than –50 cm. of water resulted in the formation of a sanguineous exudate. The formation of bronchial fistula is also to be feared from a high negative pressure.

The process of healing has been referred to so frequently as to require little further discussion. No attempt has been made to adapt a mathematical formula to the curve of healing. In fact, it is concluded that the period of healing may be modified, depending upon the adequacy of drainage and the completeness of pulmonary expansion. It is not necessary, as Roser thought, that the pleural adhesions form at the hilus and extend peripherally. They may apparently form wherever the inflamed pleurae come in contact. In rabbits encapsulation usually occurs anteriorly, in man posteriorly, the site of encapsulation presumably being influenced by gravity.

It is hardly necessary to point out that the healing of the cavity and expansion of the lung are not identical processes. Complete expansion is no assurance that the empyema is healed, and complete obliteration of the cavity implies only that the surface of the lung has been brought out to the chest wall. This latter process, however, is accompanied by pulmonary expansion if the lung is normal.

It is well known that, in empyema, the reaction of the parietal pleura exceeds that of the visceral pleura. Gay, Clark, and Linton observed as early as the third day that the thickness of the exudate on the parietal surface was greater than that on the visceral surface. In the animal experiments reported here the thickness of both pleurae varied greatly, and the disparity between the two was not so constant as is the case in clinical empyema. It has been assumed that differences in the blood supply or lymphatic drainage, possibly other factors, may explain this disparity. In the present study no satisfactory solution of this problem was encountered.

The question of what happens to the pleura after the healing of empyema may be answered with some reservations. There can be no doubt that the cavity, as a rule, becomes obliterated and that this obliteration may be permanent. Furthermore, the rest of the pleural cavity may remain completely or incompletely obliterated, at least, for a period of months or years. No evidence has been found to support the contention of Roser that the pleural adhesions eventually absorb, but observations on both clinical and experimental empyema demonstrate that in time the fibrous adhesions may become very delicate.

The assertions of Bloch and Parrish[8] that pleural adhesions may be prevented by pneumothorax and that drains inserted into the chest are the cause of crippling adhesions cannot be accepted on evidence so far presented. Experimental empyema in which pneumothorax was always induced resulted as a rule in obliteration of the pleural space on that side. Furthermore, in clinical empyema, thickening of the pleura and deformity (temporary) of the chest have been seen in patients treated by aspiration and air replacement. The most severe thoracic deformities that have been observed were in cases in which drainage was neglected.

CHANGES IN THE CELLS LINING THE ALVEOLI OF THE LUNG

In the paragraphs devoted to microscopic observations in empyema, reference was made to the development of structures in the subpleural portions of the lungs, which evidently were made up of cuboidal epithelial cells and were continuous with the cells lining the alveoli. Although this phenomenon does not appear to be directly concerned in the healing of empyema, it is of fundamental biologic importance and warrants further discussion.

A proliferation of alveolar epithelium was mentioned as a feature of experimental empyema by Coenen[25] in 1901. Hyperplasia of alveolar epithelium has also been described as a part of the pathology of several diseases of the lungs, particularly the pneumonias of measles, pertussis (Seemann[117]), and influenza (Winternitz and his coworkers[134]), also jagziekte, a specific chronic pneumonia of sheep, peculiar to South Africa (Cowdry[30]). Seemann stimulated epithelial hyperplasia by injecting staphylococci into the lungs of doves and hens, Young[135-138] by the injection of various metallic salts into the pleural space of rabbits, Fischer[37] by intravenous injection of toxic substances, and Guieysse-Pellisier[53] by exposing animals to carbon tetrachloride.

All stages of alveolar epithelial growth and desquamation have been observed. Cowdry followed small islands of proliferating tissue through in serial sections and revealed that the proliferation cells were in continuity on all sides with flattened alveolar epithelium. Winternitz, Wason and McNamara observed epithelial proliferation in influenza so extensive that it was practically indistinguishable histologically from an invasive malignant neoplasm.

It may be questioned whether the condition observed in the present study should be referred to as a true hyperplasia of epithelial (or other) alveolar cells, particularly as mitotic figures were not observed. The possibility of the cuboidal form's being due to pulmonary collapse must be given consideration. Loosli[78] was unable to demonstrate an epithelial lining of the respiratory portion of the lung of rabbits after collapsing the lungs for periods of 2 to 40 days by means of pneumothorax. On the other hand, Dogliotti and Amperino[32] observed in lungs compressed by

pneumothorax that some of the collapsed alveoli were lined with cuboidal epithelium. The occurrence of this change in the specimens reported here, only in the subpleural region where the inflammation was most intense, suggests that it is a reaction to the infection, that it represents a hyperplasia comparable to that described in the pulmonary diseases mentioned rather than an effect of compression of the lung.

No reference has been found in the literature to the occurrence of subpleural changes of this type in empyema in man. The one human case referred to here indicates that this phenomenon may occur in clinical as well as in experimental empyema.

The observations described supply further evidence that the alveolus of the lung is actually lined with epithelium. This old conception of the histology of the alveolus has recently received support from Miller,[90a] and Bremer.[12] On the other hand, Fried,[39] and Watson[131] are among those who have presented evidence in support of the idea that cells lining the alveolus have phagocytic properties and are derived from reticuloendothelium rather than epithelium. It must be granted that the evidence presented here in favor of the epithelial nature of these cells is based purely on morphology; no experiments were undertaken to study phagocytic properties.

METHODS OF TREATMENT

It is not the purpose of this article to propose that empyema be treated by a routine method. One method may be more satisfactory than another depending upon the stage of the disease or the nature of the exudate.

Aspiration is of value early, not only because it is a simple means of providing drainage and at the same time of avoiding open pneumothorax, but because by removing exudate it permits reexpansion of the lung and the formation of visceroparietal pleural adhesions. In this manner a massive empyema may sometimes be converted rather quickly into a much smaller encapsulated one. Complete healing may occur after one or several aspirations, but a routine use of this method has been found in some cases to prolong the period of sepsis, the period of healing, and the period of hospitalization.

The closed intercostal catheter method may be employed when it is desired to provide continuous drainage of fluid which is thin enough to pass through the catheter. This method combined with irrigations may be successful even in the presence of thick pus, but plugging of the tube by fibrin frequently makes the method ineffectual or troublesome.

Rib resection is of value so far as it insures more adequate drainage. The consequences of open pneumothorax can be avoided by postponing operation until the mediastinum has become stabilized as evidenced by the presence of thick pus. When performed under proper indications the mortality is as low as that with other forms of drainage. The chief objection to open drainage is that the lung is not maintained in a position of expansion. In relatively small encapsulations this is no serious objection as the lung quickly reexpands as the cavity becomes obliterated. If open thoracotomy, however, is performed for drainage of a massive empyema, healing may be prolonged. Furthermore, in the ex-

perience of Schildt,[116] open drainage of large cavities is a prominent factor in the causation of chronic empyema.

Rib resection combined with negative pressure* was advocated by Perthes[99] as early as 1898. This method has been employed more recently by Foster,[38] Muller,[91] Panton,[98] and Douglas[34] with excellent results. Heller,[62] Alexander,[1] Klages,[72] and Nicoll,[94] among others, instead of actively applying suction to the drainage tube, have attempted to maintain closed drainage by means of a water seal, a Thiersch valve, or a flap operation.

In the cases reported here a large tube and constant negative pressure provided, as a rule, complete drainage and satisfactory expansion of the lung. This method is not recommended in the incipient stages of empyema. Aspiration should be continued until it is safe to open the chest without reliance upon artificial means of providing differential pressure.

This method has definite advantages when surgical drainage is indicated in large empyema cavities containing pus rich in fibrin. The period of healing of such cavities appears to have been definitely shortened as a consequence of expansion of the lung to the chest wall, contact of the opposed pleural surfaces making possible the formation of adhesions and rapid reduction in the size of the cavity.

CONCLUSIONS

1. In the presence of open drainage, expansion of the lung occurs chiefly as a consequence of progressive adhesion of the opposed pleural surfaces. The reexpansion gained by blowing exercises is not maintained when normal respiration is resumed.

2. The application of continuous negative pressure to the drainage tube is an effective means of expanding the lung.

3. The differential pressure required to expand the lung is dependent chiefly upon the resistance offered by the thickened visceral pleura. Pressures of –10 cm. to –30 cm. of water continued for several days, usually suffice.

4. Adhesions may form at any point where the lung is expanded to the chest wall.

5. Complete or incomplete obliteration of the pleural space occurs outside the zone of encapsulation.

6. Healing, as a rule, implies complete obliteration of the empyema cavity regardless of the method of drainage employed.

7. The rate of healing is determined largely by the infecting organism, the stage of inflammation of the pleura, the size of the cavity, the adequacy of drainage, and the completeness of pulmonary expansion.

*Other forms of treatment which may be regarded as modifications of closed or open drainage, such as tidal irrigation (Hart,[55]) or intrapleural packing (Connors,[21]) are not discussed because of limited experience with these methods.

8. The fibrous adhesions remaining after obliteration of an empyema cavity become more delicate in time but have not been observed to disappear.

9. Reexpansion of the lung must be regarded as one of the fundamental principles in the treatment of acute empyema. It may be attained by the proper application of several methods of treatment.

10. Large empyema cavities containing thick pus are best treated by rib resection, insertion of a large drainage tube, and maintenance of continuous negative pressure.

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DIAPHRAGMATIC HERNIA IN INFANTS UNDER ONE YEAR OF AGE TREATED BY OPERATION*

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CLINICAL recognition of diaphragmatic hernia in the newborn and early infancy is quite unusual. The condition is so uncommon that the average physician sees it but few times during an entire lifetime. It is not strange that physicians have not recognized these cases, since as late as 1921 only 44 cases of diaphragmatic hernia had been recognized clinically, as recorded by Carman and Fineman.[1] The clinical recognition and the surgical treatment are, therefore, quite recent developments in medicine.

Since diaphragmatic hernias may exist at birth, it is very important that they be recognized by the physician and treated surgically if symptoms are produced by the condition. The occurrence of cyanosis during the newborn period or later should call for a physical examination of the chest and a roentgen film, in order that cardiac displacement and visceral herniation through the diaphragm may be discovered at the earliest opportunity.

The following report of the successful surgical treatment of diaphragmatic hernia in an infant twenty-seven days old supports the above conclusion.

CASE REPORT

Infant, J. P., was admitted to the University of Kansas Hospital on May 21, 1934. His parents gave a history of a normal delivery followed by cyanosis and increased respiratory rate which was first noticed on the seventh day of life. After the seventh day he had frequent attacks of cyanosis which appeared to be initiated or increased by crying. He regained his birth weight in about eighteen or nineteen days, but when admitted to the hospital on the twenty-fifth day of life he was slightly below his birth weight of seven and one-half pounds.

Examination showed that the child was fairly well nourished. He was quite cyanotic with rapid and labored respirations. There was dullness on percussion over the right chest, and the breath sounds were increased over the left chest. The heart sounds could be heard more plainly in the right chest. The x-ray film showed the cardiac shadow almost completely in the right thoracic cavity. Gas was seen in the left chest. The stomach emptied normally. X-ray studies visualized the entire small intestine and about one-third of the colon in the left thoracic cavity (Fig. 1). The sunken abdomen gave the appearance of unnatural emptiness.

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A combined thoracic and abdominal operation was done. A nitrous oxide oxygen ether anesthetic was used with closed mask and slight positive pressure. Incision was made in the eighth interspace extending forward from the posterior axillary

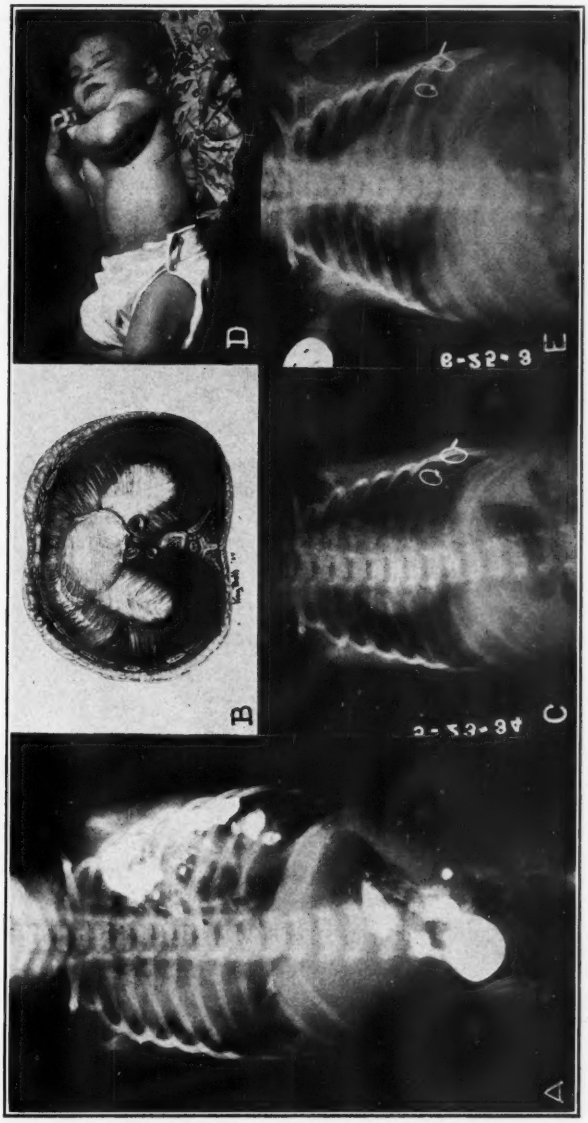

Fig. 1.—*A*, Showing barium- and gas-filled small and large intestine in left chest. Heart shadow extending far into the right chest.
B, Diaphragm viewed from above showing congenital defect in left lateral margin.
C, Radiograph four hours after operation. Heart is already returning to the normal position.
D, Photograph of baby four months after operation. The chest and abdominal scars are visible.
E, Radiograph taken one month after operation showing normal expansion of left lung and normal position of heart. The silver wire sutures were removed after fourth week.

line about 12 cm. The ninth rib was cut near its junction with the costal cartilage. The entire left chest proved to be filled with small intestine and a portion of large intestine. A triangular opening was found in the left lateral portion of the di-

TABLE I

DIAPHRAGMATIC HERNIA IN CHILDREN UNDER ONE YEAR OF AGE TREATED BY OPERATION

NAME	NO. OF CASES	AGE	SEX	OUTSTANDING SYMPTOMS	OPERATIVE TREATMENT	LOCATION OF HERNIA	RESULT
McCleave[2]	1	3½ months	M	Pain, dyspnea, cyanosis.	Anesthetic not recorded. Abdominal incision.	Left posterior	Died
De Buys[3]	1	4 months	F	Colic, unable to swallow or vomit, labored respirations, obstruction.	Ether. Abdominal incision.	Left posterior	Died
Barnett[4]	1	8 months	?	Cyanosis, abdominal distention, obstruction, vomiting.	Anesthetic not recorded. Abdominal incision.	Left—absence left dome	Died
Woolsey[5]	1	35 days	F	Dyspnea, cyanosis.	Anesthetic not recorded. Abdominal incision.	Left posterior	Cured
Olmstead[6]	1	6 months	F	Dyspnea, cyanosis, loss of weight.	Ether. Abdominal incision.	Left central	Died
Meyer[7]	1	Few weeks	M	Vomiting, obstructive symptoms.	Operation not completed. Peritonitis due to obstruction and gangrene.	Not recorded	Died
Bettman and Hess[8]	1	3½ months	F	Cyanosis, pain.	Ethylene, CO_2 pressure thoracotomy and abdominal.	Left lateral	Cured
Donovan[9]	1	4 months	M	Shortness of breath, convulsions, pain, cyanosis.	Anesthetic not recorded. Abdominal incision.	Left posterior	Cured

TABLE I—CONT'D

NAME	NO. OF CASES	AGE	SEX	OUTSTANDING SYMPTOMS	OPERATIVE TREATMENT	LOCATION OF HERNIA	RESULT
Bettman and Hess[10]	1	9 months	F	Colic, respiratory embarrassment.	Ethylene oxygen ether, pressure anesthesia. Thoracic and abdominal incision.	Left central	Cured
Eggers[11]	1	6 weeks	F	Dyspnea, cyanosis, pain.	General anesthesia. Abdominal incision.	Large left eventration	Died
Robb[12]	1	7 weeks	M	Dyspnea, cyanosis, choked when nursing.	Anesthetic not recorded. Abdominal incision.	Left posterior	Cured
Johnson and Bower[13]	1	41½ hours	?	Dyspnea, cyanosis, and obstruction.	Ether. Abdominal incision.	Left—dome	Cured
Unshelm[14]	1	9 months	M	Vomiting, malnutrition, anemia.	Chloroform-ether mixture. Abdominal incision.	Esophageal hiatus	Died
Barrett and Wheaton[15]	1	4½ months	M	Vomiting, cyanosis, constipation.	Open ether. Abdominal incision.	Left	Died
Coryllos[16]	1	13 days	F	Cyanosis, dyspnea, vomiting.	Oxygen-ether, intratracheally. Intratracheal, thoracotomy and abdominal.	Left posterolateral	Cured
Iizuka and Sano[17]	1	8 months	F	Dyspnea, cyanosis, vomiting.	Local anesthesia. Abdominal incision.	Left posterolateral	Cured
Orr and Neff	1	27 days	M	Dyspnea, cyanosis, and emptiness of abdomen.	Nitrous oxide oxygen, with positive pressure. Combined abdominal and thoracic incision.	Left lateral	Cured

aphragm with the apex pointing toward the central tendon. This opening was about 4 cm. by 4.5 cm. in size. The large intestine was adherent to the posterior margin of the opening. Attempt at reduction through the chest wound was unsuccessful. A small opening was then made in the abdominal wall just below the costal margin in the anterior axillary line about 3 cm. long. With two fingers inserted in the abdominal wound an assistant easily reduced the intestines by traction from below. The point of attachment of the colon to the posterior margin of the opening was not disturbed. The apex of the opening was closed with a double row of silk sutures for a distance of about 2 cm. This left a lateral opening which was closed by a double row of silk sutures attaching the margin of the opening in the diaphragm to the chest wall. The left lung was apparently completely collapsed, and the heart was pushed to the right beyond the midline. The chest wound was closed with plain catgut in the muscles, two small wire sutures placed to draw the eighth and ninth ribs together, and silk in the skin. Just before the wound was completely closed, the lung was partially expanded by positive pressure. The abdominal wound was closed with catgut and silk. The child left the table in apparently good condition. About twelve hours after the operation, he again became cyanotic and artificial respiration was used for a short time. During the night he was cyanotic several times, and heart action grew weak and irregular. He recovered from these attacks and four days after operation was apparently out of danger. He was sent home at the end of two weeks and returned in one month at which time the wire sutures were removed. Since the operation, the patient has done exceptionally well and now, one year after operation, is developing normally without any evidence of a recurrence of the hernia.

A review of the literature indicates that there have been sixteen infants under one year of age who have been operated upon for diaphragmatic hernia (Table I). To this number is added the case here reported. It is noted that in this group 7 were males, 8 were females and in two cases the sex was not recorded. The mortality rate has been 47 per cent. This is higher than the average as given by Hedblom[18] for all diaphragmatic hernia operations up to 1925, which was 33.6 per cent.

The outstanding symptoms in this group of cases have been pain, dyspnea, cyanosis, and vomiting as evidence of obstruction or partial obstruction. In observing these patients it has been noted in some cases that the symptoms were ameliorated by holding the infant in an upright position. The diagnosis has been confirmed by the x-ray evidence of portions of the gastrointestinal tract within the thoracic cavity.

The operative technic has varied. As anesthetics, ether, ether chloroform mixture, nitrous oxide oxygen ether, ethylene, ethylene oxygen ether, and local anesthesia have been used. In only four recorded cases is there a statement that positive pressure anesthesia was used. While it may have no significance, it is interesting to note that these four patients all recovered. It is probable that a closed method of anesthesia administration with some positive pressure is the safest. The incisions have been abdominal, thoracic or a combination of the

two. The thoracic incision undoubtedly gives the best exposure and is probably no more hazardous than the abdominal. Combined incisions seem to make reduction easier in some cases, especially if there is strangulation, incarceration, or adhesions. The negative pressure in the thorax may be a troublesome factor when attempting to reduce a hernia through an abdominal incision. In our case, we found it extremely difficult to reduce the many loops of small intestine from above, but, as soon as a small opening was made through the upper abdominal wall, the intestines were withdrawn into the abdomen quickly and without difficulty.

CONCLUSIONS

A study of the recorded cases of diaphragmatic hernia in infants under one year of age seems to warrant the following conclusions:

1. Since this type of congenital hernia is recognizable in the earliest days of life and is a menace of the most serious nature, prompt restoration of normal conditions within the thorax is desirable.

2. The operative indications for diaphragmatic hernia in infancy do not differ from such indications later in life.

3. The death rate in infants following operation has been greater than the mortality recorded for all ages.

4. Some form of positive pressure anesthesia is preferred.

5. Combined thoracic and abdominal incisions will frequently reduce the technical difficulties incident to reduction of the hernia and closure of the diaphragmatic defect.

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TUBERCULOUS TRACHEOBRONCHITIS*

Report of a Case

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INTRODUCTION

TUBERCULOUS stenosis of the trachea as a cause of death is uncommon. In the following case, death was due to an obstruction involving the trachea from its bifurcation to the thyroid cartilage. This was essentially a mural obstruction (v. Schroetter) of the mixed type, since ulceration, hyperplasia and shrinking were all present. McConkey[1] recently reported a case almost identical. For an exhaustive review of the entire subject the reader is referred to Eloesser.[2, 3]

REPORT OF CASE

History.—The patient, a white female, aged twenty-six years, was admitted May 10, 1932. She had been ill since July, 1931, at which time a diagnosis of pulmonary tuberculosis was made. Her chief symptoms were cough and expectoration. She had pleurisy frequently in the right side. She weighed 128 pounds and was in good general condition.

Physical Examination.—The patient was well nourished and well developed. Examination was negative except the chest. In the right lung breath sounds were diminished anteriorly over the second and third interspaces. Crepitant râles were audible posteriorly between the second and the sixth dorsal spines. The left lung was negative.

Laboratory Findings.—The sputum was positive for tubercle bacilli. Other findings were irrelevant.

X-Ray Report.—The bony cage was symmetrical. Left heart border normal. Right border obliterated with the density of the right lung. The trachea was deviated to the right. Left diaphragm was smooth. Right diaphragm was elevated. Border indistinct. Left Lung: Hilum contained a few dense areas of calcification. A Ghon's tubercle was present in the base over the diaphragm. The lung field was otherwise negative. Right Lung: The hilum was matted. Dense infiltration extended from the hilum into the apex and down to the diaphragm. Mottled infiltration extended throughout the remaining lung.

Subsequent Notes.—Pneumothorax was instituted in November, 1932, but a good collapse was not secured because of an adherent pleura. The patient developed fluid after a time and had an elevated temperature and pulse rate. The fluid was controlled by aspiration and the pneumothorax continued until August, 1933. On August 11, 1933, an upper stage, posterior thoracoplasty was done, air having been aspirated several times before operation. She recovered immediately from the effects of the operation. Progress notes at this time read as follows: "Shortly after the operation she had considerable trouble in breathing, and it sounded (stridor) as if she had an obstruction in the trachea, or one of the large bronchi. Her breathing at any time could be heard outside the room. At no time did her condition become alarming, however. It was decided, if necessary, to do a bronchoscopy."

While sitting up for an x-ray shortly after this the patient coughed up a large piece of hard, dried sputum. Breathing was much easier after this. The films

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showed the trachea retracted sharply to the right just below the clavicle, and it was thought at the time that tenacious sputum accumulating at this point was the cause of the stridor.

The temperature a month after operation was elevated to 100-101°. The sputum was positive. A second-stage thoracoplasty was done September 28, 1933, from which she made an uneventful recovery. The patient was discharged November 16, 1933. Sputum, negative. Weight, 125 pounds. No respiratory difficulty. The patient was seen again July 22, 1934, at which time she weighed 122 pounds. Coughed occasionally; raised very little sputum, which was negative for tubercle bacilli; doing housework.

Fig. 1.—Posterior view of heart lung specimen. (1) Line of demarcation between normal lung bronchus and diseased trachea. (2) Emphysematous, but otherwise normal left lung. (3) Right bronchus from which disease originated. Note heavy proliferations on trachea. The trachea was 6 to 8 mm. in thickness and was flattened in the A-P diameter. (4) Collapsed right lung, result of thoracoplasty.

The patient was seen again August 27, 1935. She was complaining of having bad attacks of dyspnea which alarmed her greatly. She was readmitted at this time. Her general condition was good. Weight, one hundred twenty-five pounds; no fever; slight cough. Her respirations were stridorous, and a loud wheeze was audible over the trachea anteriorly and posteriorly. Breath sounds over the right lung field were absent. The percussion note was flat throughout.

Stereoscopic x-ray pictures showed a total collapse of the right lung and a trachea which was pulled far to the right. Although somewhat narrowed, the

trachea appeared to be unobstructed and the lumen patent. Bronchoscopy was again considered, but it was thought to be too dangerous a procedure in the light of the retracted trachea. The sputum was positive. As long as the patient was quiet, she experienced no discomfort. Any exercise or excitement, however, would greatly augment her respiratory difficulty. Her condition did not become alarming until September 19, 1935, at which time breathing became very difficult. A loud wheeze was audible on both inspiration and expiration many feet from the patient's bed. She was relieved temporarily by morphine and adrenalin. On September 20 she was placed in an oxygen tent. The patient died the following day of asphyxiation.

Autopsy Findings.—Left Lung: The left lung was normal except for some emphysema. Right Lung: The lung was adherent throughout and was quite small and solid and was shaped like a small sausage. The pleura was thickened. Firm, round, yellow, caseous nodules were present throughout and were surrounded by fibrous tissue. These nodules were about the size and shape of cherry seeds and could be peeled out of their fibrous capsule. There was a great amount of fibrosis throughout, and at no point could air be expressed. The blood vessels were narrowed or obliterated.

The right main bronchus was patent, however, and was somewhat dilated in its terminal portion. Its walls were greatly thickened, and its mucous surface was destroyed. Gross proliferations and a thick, yellow exudate filled its lumen, and a probe could barely be admitted at the junction with the trachea.

The trachea was retracted toward the right lung. It was flattened in an anteroposterior direction and was 6 to 8 mm. in thickness. On opening the trachea the organ was found to be completely occluded by inflammatory tissue. No normal mucosa was visible. Fig. 1 shows the gross pathologic condition.

Microscopic Pathology.—Sections of the trachea and bronchus showed an absence of epithelium which was replaced by a dense, thick, layer of fibrous tissue. Hyalinization was visible at many places. Near the edge of the progressing lesion tubercle formation was visible with the characteristic epithelioid and giant cells surrounding the caseous centers. The lung showed a thickened vascular pleura. The alveoli were either collapsed or filled with fibroblasts. No air containing spaces were visible. Caseous masses of varying size were encapsulated with bands and whorls of fibrous tissue. None of the early cellular elements characteristic of the tubercle were present. Accumulations of carbon were visible in the lymph spaces. The blood vessels were narrowed but numerous.

SUMMARY

We failed to consider seriously tracheal tuberculosis as a cause of the obstruction, because the embarrassment of respiration was periodic. Nervousness would always create a stridor, but the patient experienced little respiratory distress until two days prior to death. Autopsy demonstrated that thoracoplasty had not retracted the trachea to a degree which would cause obstruction. Our hesitancy in bronchoscoping the patient was not justifiable. We conclude that all patients with a satisfactory lung collapse, who have a positive sputum, should be bronchoscoped.

REFERENCES

1. McConkey, M.: Occlusion of the Trachea and Bronchi by a Tuberculous Process Complicating Pulmonary Tuberculosis, Am. Rev. Tuberc. 30: 307, 1934.
2. Eloesser, L.: Bronchial Stenosis, J. THORACIC SURG. 1: 194, 1931.
3. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis, Am. Rev. Tuberc. 30: 123, 1934.

A NEW THORACOPLASTY SCREEN*

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A NEW thoracoplasty screen has been devised in the chest department of Barnes Hospital because of the inadequacies and deficiencies of the screens now in use.

The new screen is constructed of one-quarter inch copper rods and heavy wire woven into one-half inch meshes, which is found fully substantial and still not too heavy for practical purposes. The base to which the metal rods are attached is a steel strip one-fourth inch by three-fourths inch, which slips into the sliding metal attachment on either side of the operating table. The perpendicular upright rods are seven-

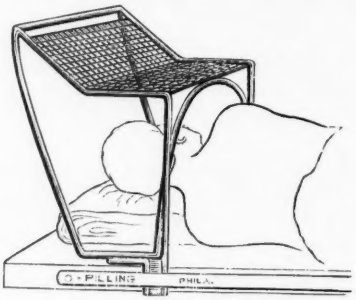

Fig. 1.

teen and one-half inches in height. Another rod arched between these supports gives additional strength. The wire shelf extends off at right angles to the uprights for six and one-half inches and then continues for another six inches at a thirty degree angle to the base. The frame is elongated two inches to allow ample room for clamping the drapes and to add height to the screen. As the screen angles upward it increases two inches in width to allow the anesthetist additional room. The reenforced wings of the screen have been designed at an oblique angle to the base to enable the anesthetic apparatus to be brought to a more advantageous position.

In practical application, the new screen allows enough room for the surgeon and his second assistant in the space limited to them on the one side of the operating table. Previously, while the surgeons worked in

*The screen is made by the G. P. Pilling & Son Co., Philadelphia, Pa.
†From the Medical and Surgical Chest Service of Barnes Hospital.
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the upper thorax the second assistant would either have to back from the operative field or be pushed against the anesthetist, thereby contaminating himself. In order to expedite the operative procedures and to secure hemostasis as quickly as possible, the second assistant must

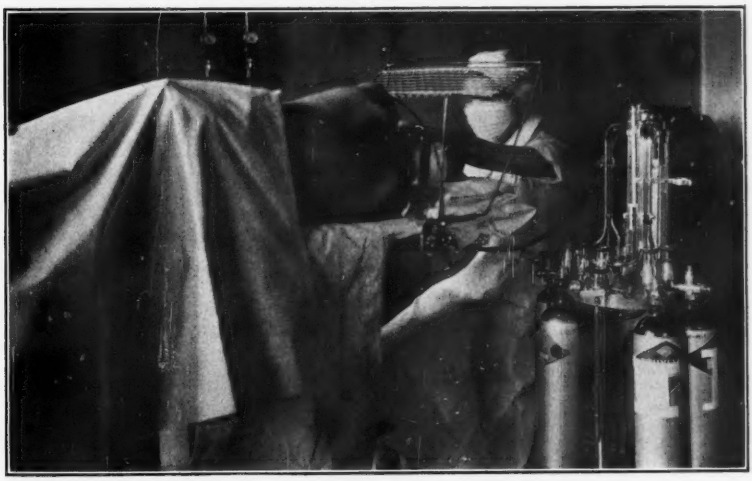

Fig. 2.

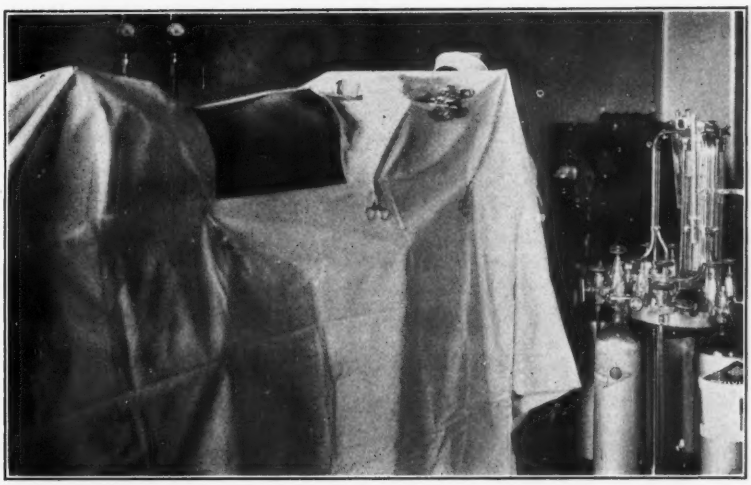

Fig. 3.

clamp vessels, sponge, and retract as the surgeon is working anteriorly. Necessary instruments are placed upon a sterile sheet on the grilled shelf of the new screen from which the second and third assistants can obtain them without removing their eyes from the operative field. This

grilled shelf also allows these assistants a place upon which to rest their arms without continually disturbing the anesthetist with poking elbows, and the wings of the screen protect the assistants from contamination by the anesthetist. As the screen allows the second and third assistants to advance to the head of the table, the surgeon and his first assistant are allowed more freedom of movement.

In summary the advantages of the new thoracoplasty screen are:

1. It eliminates chances of contamination of instruments, operative field, and the operating team.
2. It facilitates speed of operation.
3. It allows for free movement of operating team and anesthetist.
4. It protects the anesthetist.

This screen was designed primarily for thoracoplasties; though now it is being used with the same advantages for thyroidectomies, radical breast amputations, and other operative procedures which necessitate the crowding of the surgeon, assistants, and anesthetist at the head of the table.

The author is indebted to Miss Helen Lamb, chief anesthetist of Barnes Hospital, for her helpful suggestions.

Communication

In the JOURNAL OF THORACIC SURGERY **4**: 231 (Feb.), 1935, I published an article entitled "Total Removal of the Right Lung for Bronchiectasis." The patient on whom the operation was performed developed an acute respiratory infection and I referred her to the Rhode Island Hospital, where she was admitted to the medical service of Dr. Charles Gormly. Less than twenty-four hours after admission she died, Jan. 8, 1936, three years and two months after her last operation. All efforts on the part of the House staff to obtain permission for a postmortem examination were of no avail. I succeeded, however, in obtaining a signed permit following an interview with the parents at their home. Dr. B. Earl Clark's report of the autopsy findings, which are of significance to the above mentioned publication, follows:

"When the chest plate is removed, the right pleural cavity is seen to be completely obliterated by many adhesions and masses of inflammatory tissue. The height of the diaphragm on the right, through the peritoneal cavity, is felt to be at the level of the third interspace. It seems to be pulled up by adhesions into the right chest cavity. On the left it is at the level of the fourth interspace, and the mediastinum is considerably pulled to the right so that the apex of the heart is about 1 cm. to the left of the midline and the right border of the heart only 1 cm. from the lateral thoracic wall.

"On exploration of the right pleural cavity, all adhesions are broken with great difficulty, but there are no pockets of pus. A probe is passed into the axillary sinus, and there is demonstrated a chronically inflamed fistulous tract which extends into the upper main branch of the right bronchus and in turn communicates with the trachea. The trachea shows a marked deviation to the right and is attached to the right chest wall by several masses of inflammatory tissue. In the lower part of the right pleural cavity is a mass of collapsed lung tissue occupying a rather triangular space bounded mesially by the mediastinal tissues and laterally by the elevated diaphragm, to which tissues it is firmly adherent. This is connected with the trachea by a large bronchus, evidently the main lower branch of the right bronchus. This appears to be a considerable portion of the right lower lobe and measures 11 by 8 by 5 cm. In it are seen numerous bronchiectatic cavities between which is dense, red, airless fibrous tissue.

"The left pleural cavity contains numerous fibrinous adhesions. The left lung is somewhat larger than average. It weighs 900 grams, and is not collapsed. On palpation, the lung tissue is firm and rub-

bery and does not crepitate. The pleural surface of the left lung is covered with fibrinous exudate. On section there is seen to be a consolidation of the entire lung in both the upper and the lower lobes.

"Diagnosis.—*Primary Lesion:* Lobar pneumonia (left upper and lower lobes). *Secondary or Terminal Lesions:* Septic spleen. Fibrinous pleurisy, left. *Historical Landmarks:* Pneumonectomy (right upper and middle lobes). Multiple bronchiectatic cavities (right lower lobe). Bronchofistula (right axilla)."

At the time of the last operation, Nov. 8, 1932, the entire sweep of parietal pleura was free of lung tissue which was mobilized down to a narrow pedicle at the hilus. It appeared at the time that the amputation stump presented a section through the main stem bronchus and the bronchus to the upper lobes prior to its division. In the light of the postmortem findings the stump must have consisted of a section through the upper lobe bronchi just beyond the point of division. The previous cauterizations (three) had destroyed much of the diseased lung and had obscured some of the normal landmarks. The diseased, atelectatic, lower lobe was intimately fused to the posterior mediastinal structures from which it was difficult to distinguish it in situ even with the exposure possible on the autopsy table. In addition, this remnant of lung was hidden from view by the abnormally high diaphragm, to which it was also intimately fused. Thus, at the time of the hurried operation, it was not recognized and was inadvertently left behind.

SUMMARY

1. This case, then, is an instance of a bilobectomy of the right upper and middle lobes.

2. To Dr. Cameron Haight belongs the credit, which he had given to me, of having performed the second successful pneumonectomy on record.

3. Likewise, to Dr. Richard Overholt belongs the credit of having performed the first successful total right pneumonectomy on record.

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It is required by the By-Laws that all papers to be read at any meeting of the Association shall be handed to either the Secretary or the Editor immediately after their presentation.

Nineteenth Annual Meeting of the American Association for Thoracic Surgery, Rochester, Minn.

Tentative Program

MONDAY, MAY 4

9:00 A.M. Business Meeting. Assembly Room, 4th floor (old building) Mayo Clinic.
9:30 A.M. Scientific session. Plummer Hall, 14th floor, Mayo Clinic.

The Heart, Great Vessels, and Esophagus.

1. Wounds of the Heart, Penetrating and Nonpenetrating Injuries.
   Dr. D. C. Elkin.
2. Further Data on the Production of a New Blood Supply to the Heart by Operation.
   Dr. C. S. Beck.
3. The Reduction of Cardiac Irritability by the Epicardial Application of Drugs as a Protection in Cardiac Surgery.
   Dr. F. M. Mautz (by invitation).
4. An Experimental Study of Intrapericardial and Extrapericardial Adhesions With Reference to Operations Upon the Heart.
   Dr. R. M. Hosler and Dr. J. E. Williams (by invitation).
5. Obliteration of the Superior Vena Cava of Undetermined Origin.
   Dr. Alton Ochsner and Dr. J. Leonard Dixon (by invitation).
6. Remarks on the Anatomical Structure of the Esophagus and Its Frailties.
   Dr. William Lerche.

7. Diagnosis and Treatment of Benign Ulcers of the Esophagus With Case Report.
   Dr. H. R. Decker.
8. The Surgical Management of Congenital Atresia of the Esophagus With Tracheo-esophageal Fistula: Report of a Case.
   Dr. N. L. Leven (by invitation).
9. Bronchoscopic Aspects of Bronchial Tumors; With Special Reference to So-Called "Bronchial Adenoma."
   Dr. Chevalier L. Jackson.

12:30 P.M. Luncheon—Kahler Hotel.

2:00 P.M. Scientific session. Plummer Hall, 14th floor, Mayo Clinic.
Surgery of the Lungs and Mediastinum.

1. An Experimental Study of the Physiologic Changes Following Total Pneumonectomy.
   Dr. B. N. Carter and Dr. J. J. Longacre (by invitation).
2. Our Results With One-Stage Lobectomy for Bronchiectasis.
   Dr. E. J. O'Brien.
3. Lobectomy and Pneumonectomy in Bronchiectasis and Cystic Disease of the Lung.
   Dr. E. D. Churchill.
4. Intrathoracic Readjustment Following Total Removal of a Lung.
   Dr. W. F. Rienhoff, Jr.
5. Tumors of the Chest Derived From Elements of the Nervous System.
   Dr. W. DeW. Andrus.
6. Intrathoracic Xanthomatous Neoplasms.
   Dr. N. W. Phillips.

4:30 P.M. Address by Professor Ferdinand Sauerbruch—Plummer Hall, Mayo Clinic.
"Tumors of the Lung."

5:30 P.M. Informal reception—Clinic Library, 12th Floor.

8:00 P.M. Smoker—University Club, Kahler Hotel.

TUESDAY, MAY 5

9:00 A.M.-11:00 A.M. Scientific session. Plummer Hall, 14th floor, Mayo Clinic. Demonstrations and experimental work by Dr. F. C. Mann and his associates.

1. Studies on the Effect of Pneumonectomy.
   Dr. Albert Behrend.
2. Experimental Studies on Tuberculosis.
   Dr. William H. Feldman.
3. Mechanisms Involved in the Removal of Microscopic Foreign Particles From the Lung.
   Drs. Willis S. Lemon and G. M. Higgins.
4. Effect of Anesthetics and Other Substances on the Liver.
   Dr. J. L. Bollman.
5. Effect of Anesthetics on the Blood.
   Dr. Paul W. Searles.
6. Coronary Blood Flow.
   Dr. Hiram E. Essex.

11:00 A.M. Tour of inspection of Institute of Experimental Medicine.
(Transportation will be provided from the Clinic Building.)

12:30 P.M. Luncheon—Kahler Hotel.

2:00 P.M. Executive meeting. Board of Governors' Room, 3rd floor, Mayo Clinic.

2:30 P.M. Presidential Address. Dr. Carl Eggers. Plummer Hall, 14th floor, Mayo Clinic.

Symposium on, "Training of the Thoracic Surgeon."

1. From the Standpoint of the General Surgeon.
   Dr. Evarts A. Graham.
2. From the Standpoint of the Thoracic Surgeon.
   Dr. John Alexander.
3. From the Standpoint of the Phthisiologists and Internists.
   Dr. Edward Packard.

Suppurative Lesions of the Chest.

1. Pulmonary Abscess.
   Dr. Elliott C. Cutler and Dr. Robert E. Gross (by invitation).
2. The Treatment of Lung Abscess—Including an Analysis of 100 Consecutive Cases.
   Dr. Clyde Allen.
3. Four Cases of Resection of Calcified Pulmonary Abscess Simulating Tumor.
   Dr. Evarts A. Graham and Dr. J. J. Singer.
4. Suppurative Mediastinitis.
   Dr. Harold Neuhoff.

7:30 P.M. Annual dinner—main dining room, Kahler Hotel.

WEDNESDAY, MAY 6

9:00 A.M. Scientific session. Plummer Hall, 14th floor, Mayo Clinic.

Tuberculosis and Miscellaneous Subjects.

1. The Treatment of Pulmonary Tuberculosis by Means of Ambulatory Artificial Pneumothorax.
   Dr. J. A. Myers.
2. A Control Group for Studying the End-Results of Thoracoplasty.
   Dr. S. O. Freedlander.
3. Studies on Tuberculin Hypersensitiveness. The Relation of Hypersensitiveness to Tuberculin to the Postthoracoplasty Mortality.
   Dr. W. M. Tuttle.
4. Diagnosis, Treatment and Prognosis in Tuberculous Tracheobronchitis.
   Dr. Paul C. Sanson (by invitation).
5. Paraffin Pack and Thoracoplasty in Large Apical Cavities.
   Dr. Jerome R. Head.
6. Some Errors in Technique and Mistakes in Judgment Made in the Course of 1,000 Thoracic Surgical Operations.
   Dr. Norman Bethune.
7. Skeletal Deformities in Children Resulting From Empyema, and Methods of Prevention.
   Dr. Dewey Bisgard.
8. Operating Room Infections With Special Reference to Thoracic Surgery.
   Dr. Deryl Hart.

12:30 P.M. Luncheon—Kahler Hotel.

2:00 P.M. Clinical demonstrations: Plummer Hall, 14th floor, Mayo Clinic.

1. Primary Carcinoma of the Bronchus.
   Dr. P. P. Vinson.
2. Dry Clinics.
   Drs. Lemon, Kirklin, Camp, Harrington, Craig and Gray.

3. Congenital Cystic Disease of the Lung.
   Dr. Harry Wood.
4. Essential Hypertension: the Rationale and the Results of Extensive Sympathectomy.
   Dr. A. W. Adson.

4:30 P.M. Operative and bronchoscopic clinics. 6th floor, Colonial Hospital.
Drs. Harrington, Vinson and Gray.

Entertainment will be arranged for visiting ladies.

PAPERS TO BE READ BY TITLE

Lymphatic Obstruction: Dealing in Particular With Obstruction of the Lymphatics of the Chest. Alfred Blalock.

Study of 1,500 Cases of Thoracoplasty for Pulmonary Tuberculosis. Pol. N. Coryllos.

Difficulties of Thoracoplasty and Uncollapsible Cavities. Edward S. Welles.

Lobectomy for Uncontrolled Bleeding in Tuberculous Cavities. John C. Jones.

Pathological Conditons in or Near the Right Middle Lobe With Especial Emphasis on the Interpretation of the Lateral Roentgenogram. Donald S. King.

Resection of the Lower Third of the Scapula, Supplementing Resection of the First Five Ribs, as an Aid in Compressing Apical Tuberculous Abscesses, and in Conserving Vital Capacity. Emile Holman.

Localized Empyema Simulating Tuberculosis or Nontuberculous Abscess. W. L. Rogers.

Experimental Production of Bronchiectasis. Joseph Weinberg.

Variations in the Size, Ages and Physical Characteristics of the Bronchi in Relation to Their Ligature. C. E. Bird.

Closure of Large Tuberculous Cavities by Thoracoplasty. John B. Flick and J. H. Gibbon, Jr. (by invitation).

Conservation of the First Rib in Apicolytic Thoracoplasty. Howard Lilienthal.

The Nervous Control of Respiration. Peter Heinbecker.